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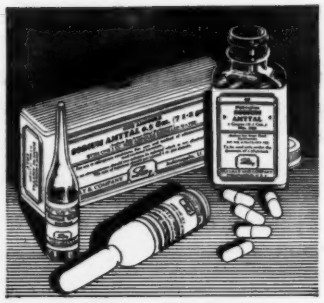

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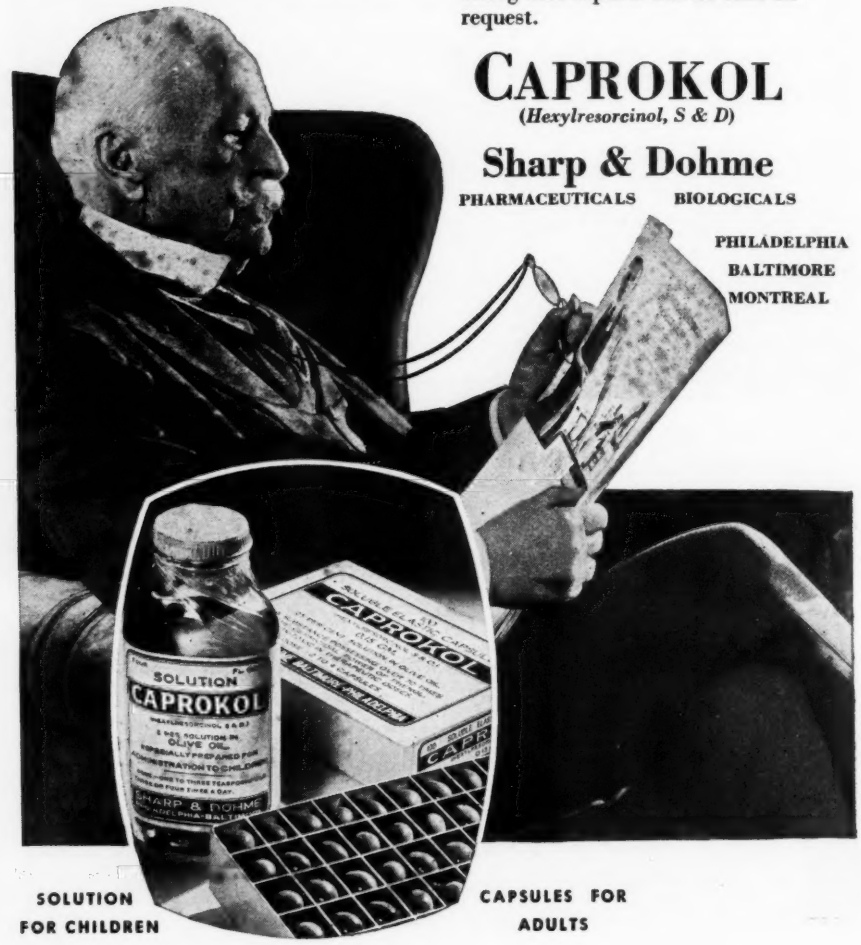

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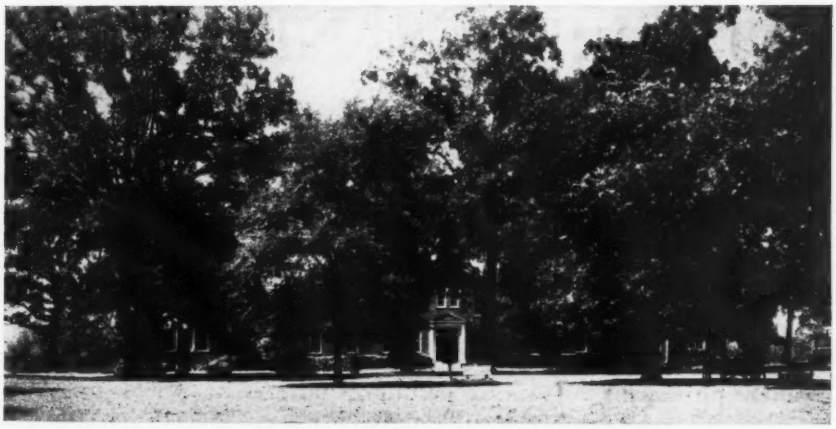

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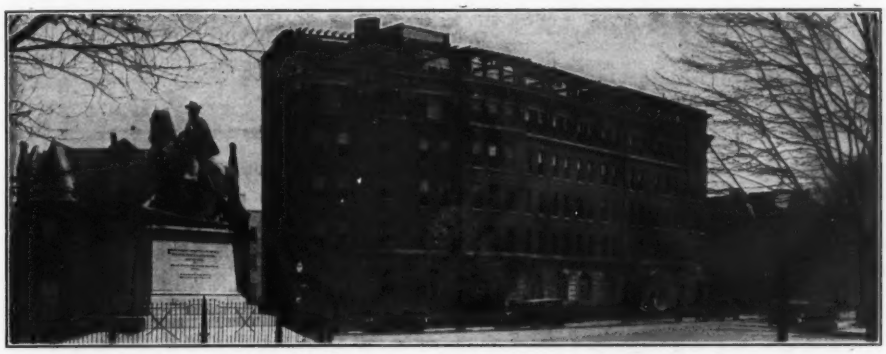

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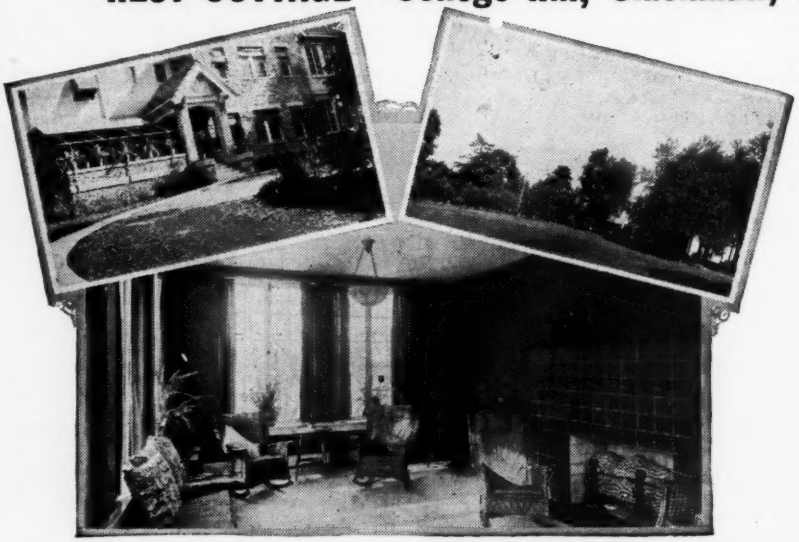

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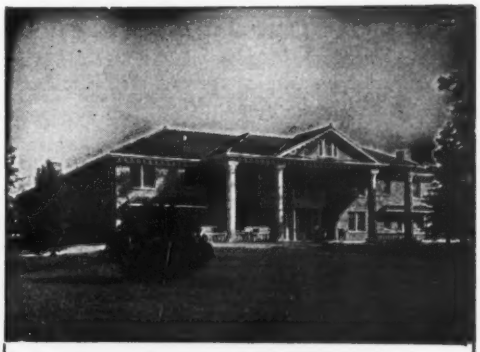

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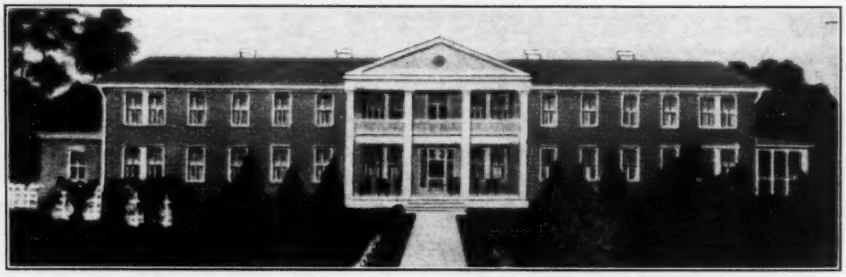

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DISEASES OF THE NAILS*

By V. PARDO-CASTELLO, M.D.,
Havana, Cuba

Affections of the nails are very common. Of 464 patients taken at randum from those attending my service of dermatology and syphilology, 192, or 41.37 per cent, had abnormal nails.

The nails are affected by the same conditions that attack the general surface of the skin, many cutaneous diseases having ungual symptoms which are nevertheless seldom characteristic. General systemic diseases such as syphilis, the endocrine disturbances, the anemias and others may also present important nail changes. The nails also suffer from conditions which are proper to them such as infections with bacteria or fungi, tumors and other inflammatory conditions. Many nail affections are congenital and knowledge of them is important in connection with the prognosis and diagnosis of these cases. Often the diseased condition of the nails cannot be traced to any general or local etiologic factor, particularly in the large chapter of the dystrophies of the nails.

It is the purpose of this paper to present from a clinical point of view some of my cases which up to the present amount to 519, and to draw some conclusions from my personal experience in the study of these conditions.

ONYCHOMYCOSIS

The most common of all diseases of the nails, onychomycosis, represents about 18 per cent of all nail affections.

Mycotic infections of the nails may be divided into onychia produced by hyphomycetes and paronychia and onychia produced by Monilia and yeasts. These two groups differ not only in their etiology, but also in their clinical characteristics. Favus is not found in my cases, since this disease is unknown in Cuba.

Parasitic onychia is always dry when caused by infection with hyphomycetes and in these cases there is absence of paronychia. The nail appears scaly, with longitudinal striations, with typical reedy appearance. It may be partially separated from its bed by corneous accumulation underneath, this separation progressing from the free edge to the root, but seldom if ever does the nail fall off. In extreme cases the nail plate is reduced to a discolored, scaly stump near the proximal end of the nail bed.

Parasitic onychia due to Monilia and yeasts is often accompanied by paronychia, and in these cases there may be some exudation. Often the primary lesion is a paronychia which is followed by distortion of the nail plate. The clinical appearance is entirely different from that described above, the lesions consisting in cloudiness and discoloration either of the outer or of the inner surface of the nail plate. This cloudiness rarely affects the whole nail, but shows as an irregular spot of creamy, yellowish, green or gray color.

Microscopical examination of filings of these infected nails show abundant fungi, and no diagnosis of onychomycosis should be made until after positive microscopical examination. The presence of mycelium or of round bodies in the nail filings or in the peri-ungual secretions, confirms the diagnosis. Cultures may be positive when there is no actual onychomycosis, since many non-parasitic fungi may be found about the nails in normal persons. Nevertheless, many fungi which are usually considered as non-pathogenic may be found in the nails in undoubted cases of onychomycosis, particularly *Penicillium brevicaule*, aspergilli and others.

In experimental work carried out by me with samples of different shoe leathers planted in Sabouraud's medium, every one gave a positive culture of an apparently non-parasitic fungus,

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but at least four of these, or very similar cultures, were obtained from several of my patients with onychomycosis. The possibility of manufactured leather being a source of infection must be considered, although I do not claim to have proved it.

Treatment of onychomycosis is disappointing in many cases. After trying several of the methods proposed by different observers, I can confidently say that strong Whitfield's ointment permanently applied has given me better results than any other procedure. Daily appli-

Condition of the Nail	Location		Race		Sex		Total
	Fingers	Toes	W.	C.	M.	F.	
Onychomycosis	44	50	64	30	68	26	94
Staphylococcic paronychia	32	4	20	16	14	22	36
Onychia traumatica	12	2	4	10	10	4	14
Unguis incarnatus (granulated)	0	4	4	0	2	2	4
Pterygium (thickened)	0	2	0	2	2	0	2
Panaritium (felon) (peri-ungual)	6	0	4	2	4	2	6
Agnails (hangnails)	6	0	4	2	4	2	6
Hematoma	4	4	6	2	6	2	8
Epithelioma	2	0	1	1	1	1	2
Bowen's disease (pinkish papule covered by horny layer)	1	0	1	0	0	1	1
Melanoma	0	1	1	0	1	0	1
Fibroma	2	1	3	0	1	2	3
Verruca vulgaris	4	0	4	0	2	2	4
Angioma (nevi)	1	1	2	0	1	1	2
Glomus tumoralis (tumor)	1	0	1	0	0	1	1
Onychatrophia (atrophy)	2	17	6	13	10	9	19
Onychauxis (overgrowth) and onychogryposis (hooked or incurved)	4	77	63	18	73	8	81
Hapalonychia (thin, soft and brittle)	2	1	2	1	3	0	3
Koilonychia (concave)	1	0	1	0	1	0	1
Onychoschizia (lamination)	1	0	1	0	0	1	1
Onycholysis (separation)	6	0	4	2	5	1	6
Onychomadesis (falling off)	0	1	1	0	0	1	1
Leukonychia striata (streaked white)	36	0	34	2	12	24	36
Leukonychia totalis (all white)	1	0	0	1	1	0	1
Onychorrhexis (splitting)	6	9	4	11	9	6	15
Beau's lines (transverse lines)	4	15	15	4	6	13	19
Dystrophia mediana canaliformis (canal-like median defect)	1	0	1	0	0	1	1
Pigmentations	11	0	1	10	8	3	11
Onychophagia (biting)	18	0	6	12	12	6	18
"Usure des ongles" (wearing)	3	0	1	2	3	0	3
Hippocratic nails (thickened curved nail, systemic)	6	6	2	4	5	1	6
Psoriasis	28	4	20	8	20	8	28
Eczema	6	18	14	10	16	8	24
Lichen planus	1	1	0	1	0	1	1
Dermatitis arsenicalis	2	2	1	1	1	1	2
Dermatitis exfoliativa	1	0	1	0	1	0	1
Radiodermatitis	2	0	2	0	2	0	2
Keratoderma punctata (with horny points)	1	0	0	1	1	0	1
Epidermolysis bullosa (loosened with deep bullae)	2	2	1	1	2	0	2
Dermatitis repens (after injury, streptococcic)	2	1	2	1	1	2	3
Scleroderma	1	0	0	1	0	1	1
Symmetric gangrene	0	3	2	1	3	0	3
Elephantiasis	0	2	2	0	1	1	2
Syphilis	10	6	13	3	11	5	16
Leprosy	15	3	10	8	16	2	18
Lupus vulgaris	1	0	1	0	1	0	1
Verruca necrogenica	2	0	2	0	2	0	2
Anemia anachlorhydria	1	0	1	0	1	0	1
Avitaminosis(?)	1	0	0	1	0	1	1
Dysendocrinias	1	1	0	2	0	2	2
Congenital ectodermal defect	1	0	0	1	1	0	1
Micronychia	1	0	1	0	0	1	1
							519

Fig. 1
Onychomycosis. Typical worm-eaten and reedy appearance of nail plate.

cations of the ointment under a finger cot must be continued for several months if success is to be expected. Filing off the diseased parts helps materially to shorten the course of the disease by removing mechanically a great deal of the infected nail. Roentgen rays are a useful adjunct to treatment in most cases.

In Monilia infections applications of tincture of iodine, together with mechanical removal of the infected material and disinfection of the peri-ungual sac, are as a rule sufficient. The source of infection should be eliminated.

The fungi found in my cases of onychomycosis were *Epidermophyton inguinale, Trichophyton interdigitale, Trichophyton niveum* and a fungus similar to *Trichophyton purpureum*. Aspergilli, Penicillium and others were also found. *Monilia albicans* and an orange-colored *Monilia,* unclassified, were also cultivated.

Onychomycosis coexists with dermatophytosis of the feet in about 30 per cent of my cases. Dermatophytids of the hands may be the cause of a chronic onychia with secondary staphylococcic paronychia. These cases are the result of nutritional disturbances produced by the neighboring inflammation and no fungi are ever found in these nails.

BACTERIAL ONYCHIA AND PARONYCHIA

Bacterial infection of the peri-ungual tissues and consequent disturbances of the nail plates are rather common, representing about 7 per cent of all nail diseases. In all of my cases infection was due to *Staphylococcus albus,* but cases due to colon bacillus infection have been reported by Hollander,[1] Jaubert[2] and one case due to tubercle bacillus by Stokes.[3]

Most of these cases are chronic, but they may present acute phases with or without the formation of a panaritium. They occur in women more often than in men, usually in cooks, laundresses, charwomen and also in bartenders and others who keep their hands wet for long periods.

One, or more often, several finger nails are affected, showing redness and swelling of the peri-ungual tissues with purulent discharge from the nail grooves. The nail plate may be distorted, scaly, broken and sometimes may fall off, to be replaced later.

The differential diagnosis with onychomycosis must be made by microscopical examination and by cultures, particularly to differentiate them from cases due to Monilia and yeasts.

The treatment of these cases requires keeping the hands dry and sometimes a change of occu-

Fig. 2
Monilia infection of the thumb nail. Typical cloudy spot affecting under surface of the nail plate.

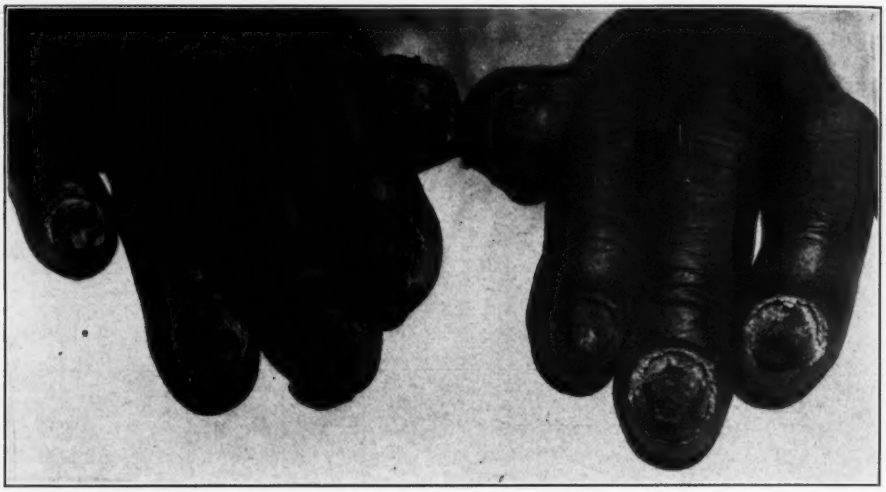

Fig. 3
Onychia and paronychia produced by yeast.

pation may be necessary if permanent results are to be obtained. Local antiseptic preparations are as a rule ineffective. The best treatment in my hands has been the daily application of a 5 per cent suspension of chrysarobin in chloroform as recommended by Morrow and Lee. Gentian violet in 5 per cent solution is also useful. Autogenous vaccines may be of help in some cases, particularly in those due to the colon bacillus.

DYSTROPHIES OF THE NAILS

Onychauxis and onychogryposis are the most common of the nail dystrophies. Hypertrophy and distortion of the nails are usually found in the toes and very rarely in the fingers. The nails may be slightly enlarged either in thickness or in width or both at the same time, or they may be enormously distorted in several shapes, sometimes resembling a horn or a hook, grayish or black in color.

The etiology of this condition varies and often no apparent cause can be found. Sometimes senility, ill-fitting shoes, dirt and neglect may cause nail hypertrophy. It may be observed also in cases of tabes, peripheral neuritis, and other nervous diseases and in trophic disturbances. Onychauxis has been described in dystrophia adiposo genitalis and in other diseases of the ductless glands.

Onychatrophia is not rare in the toes and it is due in most cases to tight-fitting shoes. In practically all civilized peoples the nails of the small toes are atrophic and sometimes almost absent. Atrophic nails are of small size, lusterless, thinned and do not grow to form a free edge. Sometimes the nails are represented

Fig. 4
Paronychia and staphylococcic onychia in a dish washer.

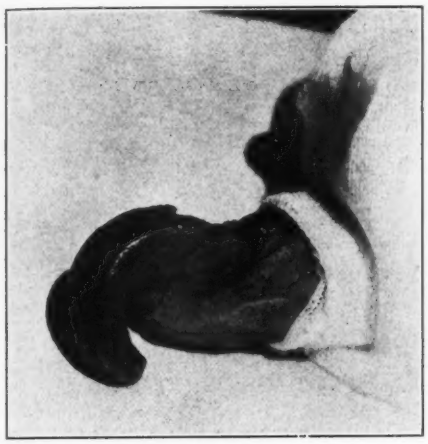

Fig. 5
Onychogryposis in a case of senility.

by bands of corneous matter more or less heaped up toward the center. I have seen two cases of atrophy of the nails in hyperthyroidism. Hollander[4] and Bloch[5] have reported cases of disturbances of the ductless glands with atrophy of the nails.

Hapalonychia is a rare nail dystrophy consisting in thinning and brittleness of the nail plate. The nail is translucent, sometimes longitudinally striated and, due to its softness, it may bend and break easily. The color is a peculiar pinkish white, resembling that of the shell of the egg. I have seen two cases of hapalonychia in chronic rheumatism. It is also seen in leprosy and in acro-asphyxia with excessive sweating of the hands and feet.

Koilonychia, also called "spoon nails," may affect one or several nails of the fingers. The disorder consists in the formation of a concavity in the nail plate without apparent change in color and without desquamation. It is a rare condition. It has been described in cases of syphilis by Howard Fox[6] and others, in cases of anemia by Broekema,[7] and according to Heller[8] it may be a symptom of eczema of the nails. I have seen koilonychia in a case of syphilis, but I have never seen this condition in cases of eczema.

Onychoschizia is a rare dystrophy consisting in the lamination of the nail plate in two or more superimposed layers. It is often confused in text-books and described as synonymous with onycholysis. The nails seem to grow normally until nearing the free edge, when they split into two or more layers which break off easily. There are no changes in texture or color. I have seen a case of onychoschizia in a young woman who had no other apparent disturbance except a slight seborrheic dermatitis of the scalp and face.

Onycholysis consists in the gradual separation of the nail plate from its bed, commencing at the free edge and progressing toward the root. The nail may separate almost entirely from the bed, but very seldom falls off. The nail loses its shine, becomes curved laterally, and the color turns yellow or gray. Often all the nails of the fingers and toes are affected. This process is not accompanied by inflammatory changes. I have seen cases of onycholysis in syphilis, eczema, psoriasis, anemia, and others in which the etiology could not be ascertained.

Onychomadesis consists in the spontaneous loss of the nails and affects all the toe nails and finger nails at the same time. The process begins at the root of the nail and rapidly progresses to the free edge. Some cases have been reported in which the condition was hereditary and of cyclic occurrence. I have seen this condition in hysteria, scarlet fever, epidermolysis bullosa and other conditions.

Leukonychia is one of the most common dystrophies of the nails. The punctate and striate types are very frequent in women, representing

Fig. 6
Pigmented stripe in a white man.

Fig. 7
Bowen's disease.

about 7 per cent of all my cases. Leukonychia totalis is very rare. Leukonychia seems to be due to defective keratinization in groups of cells with the presence of granules of keratohyaline, which would explain the white color. There seems to be no foundation to the belief that the white spots are due to the presence of air between the nail cells. Leukonychia has no pathological significance and is probably due to small traumas in the nail matrix.

Onychorrhexis is the longitudinal striation of the nails with or without the formation of fissures. It has been reported in cases of eczema, psoriasis, lichen planus, onychomycosis, nervous diseases and trophic disturbances of the extremities. It is frequent as a senile change without any other apparent cause.

Beau's lines are seen in the nails when for any reason the function of the matrix is impaired. The continuity of the nail growth suffers and there appear one or several transverse grooves. This groove may be so deep that it divides the nail in two portions, the distal part becoming cast off. Beau's lines may be the result of psychic disturbances, nervous shock, trauma, infectious diseases, pregnancy, eczema, psoriasis and many other etiologic agents. In medico-legal practice Beau's lines may serve to ascertain the approximate date of the occurrence of the condition which caused them, since the nail takes approximately 160 days to grow from the matrix to the free edge.

Dystrophia mediana canaliformis is a rare dystrophy described by Heller.[9] I have seen a case of this condition in a woman aged 25 who had a longitudinal groove in the nail of the middle finger of the left hand of five years' duration. The etiology of these cases is unknown. According to Heller, they may be due to a nervous disorder affecting the normal keratinization of the nail.

Pigmentation of the whole nail plate, affecting either one or several fingers or toes, is extremely rare. More common is the partial pigmentation in the form of bands or striae. In colored persons longitudinal pigmented bands in one or more fingers and toes are very common. In white persons these bands are occasionally seen and they are supposed to be due to the presence of pigmented nevi in the nail matrix. I have seen one of these cases in a young white man in whom the pigmented band had existed since birth without variation. Montgomery, Templeton, Ochs, Engman and Mook and others have reported similar cases.

Pigmentation of the nails is often seen in surgeons who use mercurial solutions for disinfection of the hands. The nails are grayish in color. I have seen a violaceous discoloration of all finger nails probably due to phenolphthalein ingestion. In chronic arsenical poisoning

Fig. 8
Eczema of the nails.

Fig. 9
Syphilitic chancre.

the nails may show dark pigmentation of the same hue as that observed in the skin. In argyria the nails may also show the typical slate blue discoloration. In onychomycosis, staphylococcic paronychia and in some ungual dystrophies, the nails may be gray, green or yellow. The nature of this pigment is not known. A dead nail always changes color and becomes black, gray or green.

Hippocratic nails and drumstick fingers and toes are seen in tuberculosis of the lungs, cancer of the lungs and in other chronic respiratory and cardiovascular diseases. They may be due to impaired peripheral circulation.

TUMORS OF THE NAILS

Tumors develop in the nail bed or in the periungual tissues. Epithelioma, melanoma, fibroma, angioma and verruca vulgaris are the growths most commonly found. Chondroma, sarcoma and glomus tumoralis are rare.

I have seen three cases of epithelioma of the nail tissues, of which the most interesting was that of a white woman aged 75 who for three years had what appeared to be an eczema of the last phalanx of the middle finger, with loss of the nail and gradual extension of the lesion to the rest of the finger. The condition consisted of an erythematous surface covered with keratosic crusts and of a fungating mass about one-third of an inch in diameter. Biopsies of both types of lesions showed the typical structure of Bowen's disease in the erythemato-keratosic parts and of prickle-cell epithelioma in the hypertrophic mass. The finger was amputated.

Melanoma is occasionally seen in the toes. I have seen a case affecting the nail groove with invasion of the nail bed of the great toe in a white man aged 75. It was a blackish tumor about the size of a hazel-nut. The black discoloration invaded the adjacent tissues and the nail plate itself.

According to Heller,[8] only 23 cases of authenticated melanomas of the nails have been reported, 12 in men and 11 in women, usually located in the great toes.

Fibroma is of common occurrence. In one of my cases the lesions began as small digitated excrescences in the posterior nail groove and had the histopathological appearance of angiokeratoma, with large vascular lakes situated under the epidermis and hyperkeratosic productions of the surface. The older lesions were typical of fibroma.

Verruca vulgaris is very common in the periungual tissues, particularly in the fingers in children and young adults. They are usually multiple and even appear between the nail plate and the tip of the finger, forming irregular masses. Roentgen ray treatment in suberythema doses is specific in these cases.

Fig. 10
Onychia superficialis ondulata in early syphilis.

UNGUAL MANIFESTATIONS OF THE DERMATOSES AND OF SYSTEMIC DISEASES

Psoriasis and eczema are skin conditions which often affect the nail. Psoriasis of the nails represents 5.38 per cent and eczema 4.61 per cent in my statistics.

The lesions of psoriasis and eczema of the nails are not characteristic. Psoriasis produces the well known onychia punctata or pitted nail, but this may also appear in eczema of the nails and in chronic rheumatism. Multiple Beau's lines, onychia squamosa, onychomadesis, onycholysis and onychorrhexis are often found in psoriasis. The most extensive cases seen by me were in two patients with psoriasis arthropatica, in which all finger and toe nails were affected. Roentgen ray is the best treatment for psoriasis of the nails.

Eczema of the nail may affect the nail plate and the peri-ungual tissues. In acute cases the first symptom is usually a discharging paronychia with secondary pus infection. In chronic cases the inflammatory condition affecting the fingers spreads to the skin around the nails and the nails themselves become distorted, scaly, and finally present onychorrhexis, Beau's lines and corneous accumulations between the plate and the bed. The nail may be shed and replaced several times. In some cases nail atrophy is the result of chronic inflammation of the matrix. In this group may be included those cases of dermatitis venenata observed in physicians, nurses, chemists, silversmiths, photographers or in allergic conditions of the hands and feet of eczematoid type. Onychauxis and onychogryposis are encountered in cases of chronic eczema of the legs in old people and in chronic leg ulcers.

Nail changes in epidermolysis bullosa are very characteristic. The constant formation of bullae in the extremities of the fingers and toes affects the nutrition of the nails. The nails become atrophied and the fingers and toes affect the form of smooth, bulbous stumps. Sometimes the nails are represented by small pieces of keratotic material embedded in the last phalanx and in some cases all trace of the nail plate disappears and even the nail bed becomes indiscernible. The resulting smooth scar is often studded with cysts of milia.

Onychodystrophies, loss of the nails, or inflammatory reactions of the peri-ungual tissues have also been observed in lichen planus, dermatitis exfoliativa, pityriasis rubra pilaris, ichtyosis, alopecia areata, acanthosis nigricans, Darier's disease, radium dermatitis, keratoderma palmo-plantaris, pemphigus, dermatitis herpetiformis, dermatitis repens, scleroderma, poikiloderma atrophicans vasculare, elephantiasis and lupus erythematosus.

Syphilis.—Syphilis may affect the nail plate, the nail bed and the peri-ungual tissues. Fragility and fracture of the nails, partial loosening of the nails, complete loss of nails and hypertrophic onychia are the most common affections. Besides these clinical forms described by Fournier, several authors have reported cases of koilonychia, onychogryposis and the so-called "scabrities unguium syphilitica" which consists in complete crumbling of the nails with the formation of stratified adherent layers. All of these types are found in the late stages of the disease.

I have seen two cases of a peculiar condition of the nails of all fingers in women suffering from secondary syphilis. It consists in the presence of elegant arabesques, of ripples and waves affecting the outer surface of the nails from the lunula to the free border. For this type the name "onychia superficialis ondulata" has been proposed.

Peri-ungual lesions are more frequent than those mentioned above. Syphilitic chancre is observed about the nails in 5 per cent of cases of extragenital chancres. It may be ulcerative or hypertrophic and may destroy the nail plate. There is a superficial erosive type of chancre which affects the skin surrounding the nail in a crescentic form (*chancre semi-lunaire de Fournier*). Often a syphilitic chancre in this location shows intense inflammation and may be confused with a staphylococcic paronychia or with a panaritium.

In secondary syphilis the nails may be the seat of peri- or subungual papules and of paronychia ulcerosa. As a rule only one or two fingers or toes are affected in these cases. The most important of these lesions is the ulcerative type, the "onychia syphilitica maligna" of some authors. The ulcers surround the nails, present a dirty, foul discharge, necrotic edges and may cause the loss of the nails and serious inflammation with necrosis of the fingers or toes. These lesions are very rebellious to treatment and it

must be kept in mind that often they are the only apparent manifestation of a relapsing and grave form of syphilis.

Syphilitic gumma may affect the nail tissues, but it is very rare in this location.

In congenital syphilis nail lesions are not unusual. Multiple inflammatory paronychias with nail loss have been reported by Heller[8] and also onychia sicca with atrophy of the nails, syphilonychia ulcerosa and congenital anonychia.

Leprosy.—In leprosy, onychauxis, onychogryposis and an extensive variety of forms of atrophy of the nails may be encountered. The nail lesions of leprosy are usually located in the fingers in connection with atrophy of the extremities. In lepra tuberosa the analgesic felon may cause paronychia and loss of nail, always painless. Flictenae in the peri-ungual skin are also seen in cases of nervous leprosy and at times are one of the earliest phenomena observed.

Anemia.—One of the most peculiar and interesting lesions I have observed in my series of nail cases was that of a man aged 66 suffering from anachlorhydric anemia. All finger nails were dry, discolored, with longitudinal striations, thinned and separated from their bed (onycholysis) in the anterior two-thirds. There was no inflammatory reaction. The toe nails were apparently normal.

Broekema[7] has reported two cases of koilonychia in patients with anachlorhydric anemia. The nail lesions disappeared after treatment for the anemia.

Avitaminoses.—In many cases of pellagra observed by me I have never seen lesions of the nails that might have been ascribed to this disease. White and Stillians (personal communication) have observed cases of "brittle, ridged, and furrowed nails, and this has cleared up on vitamin therapy, although it had resisted other methods of treatment."

Endocrine Disturbances.—In the examination of 562 patients with endocrine disturbances, Heller[10] found only 8 cases in which the possibility of ungual lesions of glandular origin could be considered. Hollander, Bloch, Lisser, Combes and others have referred cases of diverse dystrophies of the nails in connection with diseases of the ductless glands. The best known of these nail dystrophies is that found in myxedema, in which the nails are thin, brittle and discolored.

CONGENITAL AFFECTIONS OF THE NAILS

Congenital ectodermal defects often present nail changes, onychorrhexis, brittleness, atrophy or divers dystrophies in fingers and toes. In a case reported by me in a negro aged 21, all finger and toe nails were atrophic, represented by irregular bands of gray color adherent to the nail bed. Of 8 cases from the literature reviewed by MacKee and Andrews,[11] 4 represented dystrophic nail changes, 3 had apparently normal nails and in one case the condition of the nails was not described.

Complete absence of the nails or anonychia has been reported by Berge and Weisenbach.[12] Histological examination in this case showed absence of the nail matrix.

Micronychia congenita of one or more nails is not rare, particularly in the toes. Macronychia congenita is rarely seen except in some cases of syndactylia and in the very rare cases of congenital hypertrophy of the extremities or partial gigantism.

Under the name of "pachyonychia congenita" Jadassohn and Lewandowsky[13] reported a case of hypertrophy of all finger and toe nails coincident with keratosis follicularis disseminata, palmar and plantar hyperkeratosis and leukokeratosis of the tongue. Howard Fox[14] has reported a similar case.

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BENIGN TUMORS OF THE SMALL INTESTINE*

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After a brief review of the literature, the following three unusual cases will be reported: (1) cavernous hemangioma of the duodenum, (2) myoma of the ileum, and (3) multiple fibromata of the ileum.

Heurtaux,[1] in 1899, stated that the benign tumors of the intestine might arise from (a) the mucosa, adenoma; (b) the submucosa, fibroma; (c) the muscularis, myoma; (d) the fat cells of the external coat, lipoma; (e) the fibrous tissue of the serosa, fibroma; (f) the epiploic appendages, lipoma; (g) the blood vessels of the intestinal wall, angioma; and (h) possibly from the muscularis mucosae, myoma. Heurtaux's work was based on a paper by Steiner[2], which appeared during the preceding year, on myomata of the gastro-intestinal tract. Heurtaux discarded all of Steiner's cases except those studied histologically, twenty-nine in number, and to these he added three myomata.

King,[3] in 1917, published a most exhaustive article on the clinical-pathological side of benign intestinal tumors with special attention given to fibromata. His series included a study of forty-seven tumors of the small intestine, of which one was a hemangioma, twenty-two were myomata, nine fibromata, three myxofibromata, and twelve miscellaneous tumors. They were distributed as follows: duodenum five, jejunum eight, ileum twenty-three, and small intestine (not designated) eleven.

It seems that Heurtaux was influenced by Longuet[4] in his rejection of certain of Steiner's cases, while on the other hand Longuet accepted some of the tumors reported by Tédenat,[5] but for no apparent reason rejected others, although Tédenat's cases with the exception of one were studied histologically. King included these cases in his report, but rejected others as being of doubtful authenticity.

Brown,[6] in 1924, collected thirteen cases of vascular tumors of the small intestine from the literature and reported one case of his own. Since that time his pathological classification of these particular tumors has been the model for more recent writers.

Clifton and Landry,[7] in 1927, published a detailed review of the literature regarding fibromata of the intestines and reported a case of their own. They, in common with others who had previously written on the subject, included under this heading the following: fibroma, myxoma and variations between these two types; realizing the variance of opinion in classifying these together or separately as a result of the divergent views maintained by different pathologists.

Raiford,[9] in 1931, summarized the radiological aspect of eighty-two primary tumors of the small intestine from the records of the Johns Hopkins Hospital. In a later paper Raiford[10] discussed the clinical-pathological side of these tumors and of six additional cases collected in the meantime. Of the eighty-eight tumors summarized,* thirty-nine were malignant, and in forty-nine cases the tumor was benign. The classification and frequency of occurrence of the benign tumors are as follows: adenomata sixteen, fibromata four, myomata four, lipomata six, angiomata three, pancreatic rests six, hematomata two, argentaffine tumors seven, and cysts one. The tumors were distributed in the intestinal tract as follows: duodenum thirteen, jejunum ten, ileum twenty-five, and not stated one.

This relative frequency of benign tumors of the small intestine as compared with the malignant tumors in contrast to both types of the tumors in other portions of the gastro-intestinal tract is noteworthy. This low occurrence of malignancy has been explained by Fiedler,[11] quoting Bunting, Waldeyer, Birch-Hirschfield and Ransome, by the lack of epithelial points, the absence of irritation and the poor nutritive qualities of the small intestine.

Cave,[12] in 1932, reported six cases of benign tumor of the small intestine from the Roosevelt Hospital in New York City. He mentioned forty-five cases of tumor of the small intestine from a personal communication with Patterson, of the Massachusetts General Hospital, but did not state the details of these cases.

Fiedler,[11] in the same year, briefly summarized the clinical aspect of benign tumors of the small intestine, and reported a case of fibroma of the ileum causing intussusception.

Rankin and Newell,[13] in 1933, summarized thirty-five cases of benign tumor of the small intestine from the records of the Mayo Clinic, eleven of which had been previously reported. It is interesting to note that six of the remaining twenty-four cases came to operation within a nine-month period. The thirty-five tumors were distributed in the small intestine as follows: duodenum fifteen, jejunum nine, ileum seven, small intestine (not designated) two, ileo-jejunal junction one, and not stated one. Adenomata and myomata occurred with the same frequency and comprised two-thirds of the total number of the tumors. Half of the remaining one-third were fibromata. The rarer tumors included lipoma, hemangioma, cystadenoma, adenomyoma and osteochondroma. It is believed that the last mentioned tumor is the only one of its kind mentioned in the literature as occurring in the intestinal tract. Such conditions as pancreatic rests, argentaffine tumors, hematomas, cysts and endometrial transplants that have been included in other authors' series as neoplastic growths are purposely omitted by Rankin and Newell.

We, therefore, feel that the occurence of benign tumors of the small intestine is of sufficient frequency to call for a consideration of their presence in patients presenting certain symptoms and conditions, namely: intussuscep-

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*In presenting a summary of Raiford's studies only the material contained in *Tables IX* and *X* of his latest article[10] is included in this report, as these tables, furnishing the essential features of each of the eighty-eight cases, differ slightly from the less detailed *Table III* of the same paper, and from *Table I* of the article published the preceding year.

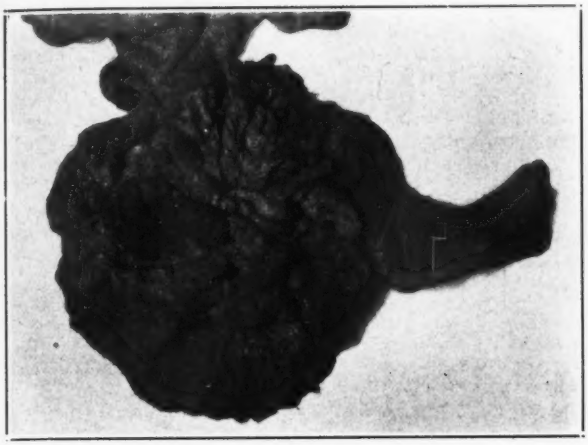

Fig. 1

Cavernous hemangioma of duodenum. The duodenum has been opened along its greater curvature, and the anterior wall of the third portion turned back to expose the tumor with its large, dark red, spongy, circular folds. Opened pylorus, above; pancreas, right. Photograph.

tion in adults, obscure intestinal disorders, and anemia of doubtful origin.

The most common clinical symptoms of duodenal tumors, malignant or benign, are hemorrhage and anemia. Kellogg and Kellogg[14] state that the anemia is often profound and may be the only symptom, that it occurs with or without ulceration of the tumor and may cause sudden death.

The same condition exists in the jejunum and ileum, but to a lesser degree, with the added probability of intussusception due to the mobility of this portion of the intestinal tract. Intussusception is believed to be due to a gripping of the tumor by peristaltic waves causing that section to be telescoped within a section lower down. Intussusception occurring in this manner differs from that in children in which it is spontaneous and thought to be due to a hyperactive gut.

CAVERNOUS HEMANGIOMA OF THE DUODENUM

Vascular tumors of the small intestine are surprisingly rare. The literature reveals twenty-four such cases.

Brown,[6] in 1924, studied thirteen cases of vascular tumors of the small intestine collected from the literature and reported one case of his own, a cavernous hemangioma of the jejunum. These tumors were distributed as follows: duodenum two, jejunum three, ileum one, small intestine (not designated) seven, and the entire intestine one. He recognized four types of the tumors: (a) multiple tumors of the vascular arcades, appearing as small red nodules in the submucosa and connected with either the veins or the arteries of the intestinal wall; (b) the submucous type which grows toward the lumen of the intestine and may ulcerate the overlying mucosa by pressure and trauma; (c) the submucosal tumor which may attain such a size as either to obstruct the lumen of the tract or cause a change in normal peristalsis resulting in intussusception; and (d) the diffuse, ring-like tumor which begins in the submucosa, involves the muscularis, constricts the lumen of the gut and results in intestinal obstruction.

Balfour and Henderson,[15] in 1929, reported a case of hemangioma of the duodenum.

McClure and Ellis,[8] in 1930, collected four additional cases of vascular tumors from the literature and reported one case of their own consisting of multiple tumors of the entire small intestine, some of which had undergone malignant degeneration.

Raiford,[10] in 1932, summarized the clinical-histological aspect of three cases of hemangiomata, one each occurring in the duodenum, jejunum and ileum from the records of the Johns Hopkins Hospital.

In October of the present year, Rankin and Newell[13] reported from the records of the Mayo Clinic a case of ulcerated hemangioma of the duodenum, causing severe gastro-intestinal hemorrhages.

Fig. 2

Cavernous hemangioma of duodenum. Note the large, irregular, endothelial lined spaces filled with blood. Photomicrograph x 200.

Of the twenty-five cases reported in the literature, including the authors' case, ten were in males and thirteen in females. The sex was not mentioned in two cases. The ages of the patients varied from two months to seventy-nine years. The tumors were distributed in the intestinal tract as follows: duodenum six, jejunum five, ileum four, small intestine (not designated) eight, and the entire bowel two. The tumors were multiple in nine instances, and in sixteen cases the tumor was single, twenty-three were intraluminar and two extraluminar.

In all cases in which any statement was made, the tumors arose from the submucosa and from this point tended to invade the muscular layer. In all cases the peritoneum remained intact. The mucosa was ulcerated in four cases, but was not involved by the tumor itself. In one case the tumor involved the mesentery; and in another the appendix was involved.

REPORT OF CASE

J. C., a negro man, aged fifty-eight years, was admitted to the medical service of the Memphis General Hospital January 16, and came to autopsy February 7, 1933, with symptoms and physical findings referable only to a severe and rapidly progressive anemia. The blood picture was that of chronic blood loss plus infection. The anemia was of a hypochromic-microcytic type.

Postmortem Examination.—The stomach was greatly dilated with a thin wall, as were the first and second portions of the duodenum. The third portion of the duodenum was enlarged, its external surface was of a dull red appearance, and instead of being soft and easily compressible it was thick-walled and of a much firmer consistency than normal. The fourth portion and the remaining intestinal tract presented nothing unusual. On section the mucosa of the stomach and first and second portions of the duodenum appeared normal. The circular folds of mucosa in the third portion of the duodenum were greatly enlarged, and projected into the lumen, forming a diffuse, annular tumor 6 cm. in width and almost constricting the lumen (*Fig. 1*). The mucosal surface of the tumor was velvety, dark red, and spongy. On section, these characteristics were limited to the mucosa and submucosa. About the periphery of the tumor, beneath the mucosa, were many small yellow areas arranged in large and small clusters. The regional lymph nodes appeared normal.

The marrow of the flat bones did not appear unusual; that of the long bones consisted for the most part of fat, but contained foci of hyperplastic marrow.

Microscopic Examination.—Section of the duodenum above the tumor revealed a normal histology. Marginal section of the tumor revealed extensive small round-cell infiltration with clustered polymorphonuclear leucocytes in all layers, but especially in the mucosa and submucosa on the one side, and a thickening of the submucosa, with leucocyitic infiltration and numerous small endothelial-lined spaces filled with lymph on the other. Section through the tumor (*Fig. 2*) revealed a structure similar to that of the tumor portion of the marginal section only more pronounced; many large endothelial-lined spaces filled with erythrocytes were present in the mucosa, but mostly in the submucosa. The muscular layers were edematous and infiltrated with polymorphonuclear leucocytes. Smaller endothelial spaces filled with lymph were also present in the tumor mass, but the large blood-filled spaces dominated the picture. Section of the regional lymph nodes revealed a normal histology except for polymorphonuclear infiltration.

Examination of the marrow of the long bones revealed a typical aplastic condition except in the hyperplastic areas. Even in these areas the marrow consisted largely of fat, but the neutrophilic myelocyte with distinct, large, dark staining granules was the predominating cell.

Sections of the liver showed enormous deposits of hemosiderin pigment throughout.

Anatomical Diagnosis.—Cavernous hemangioma of the third portion of the duodenum, with partial obstruction of the lumen of the third portion, and dilatation of the upper portions of the duodenum and stomach;

Fig. 3

Myoma of ileum. Below, external surface showing a smooth, hyaline, covering serosa. Above, sectional surface with coarse, pink fibers and ulceration of mucosa. Ileum, above tumor, dilated; below, collapsed. Photograph.

chronic duodenitis; and anemia, severe, of the hypochromic-microcytic type (clinical).

In this case of cavernous hemangioma of the duodenum, the outstanding clinical symptom was a severe and rapidly progressive anemia of the hypochromic-microcytic type.

MYOMA OF THE ILEUM

The myoma is not the most common type of benign tumor of small intestine, but it does occur with sufficient frequency to justify consideration. The literature reveals forty-six such cases.

King,[3] in 1917, summarized twenty-two cases of myomata of the small intestine previously recorded. In regard to the etiology of these tumors he accepted the theory of Longuet, who compared their rarity with the frequency of similar uterine tumors, and concluded that in the case of the uterus the muscle is the main tissue of the organ and by nature adapted to excessive development, whereas, the reverse is true of the muscularis of the intestine. He also concluded that these tumors arose from the muscularis, as there was but slight possibility that any of them had originated in the muscularis mucosae.

Rhodenberg,[16] in 1919, reported a case of myoma of the jejunum. Goldschmidt,[17] in 1923, reported a case of bleeding myoma of the jejunum. Camp,[18] in 1924, published a case report of myoma of the duodenum. Plenk,[19] in 1927, reported two cases of myomata of the intestine; one, a single tumor of the small intestine, and the other a case of multiple tumor of the ileum and cecum, in which a large tumor mass in the lower ileum showed sarcomatous degeneration.

Balfour and Henderson,[15] in 1929, reported two cases of myomata of the duodenum. Kahn,[20] in 1931, reported a myoma of the jejunum.

Raiford,[10] in 1932, collected two cases of myomata of the jejunum and two of the ileum from the records of the Johns Hopkins Hospital. In the same year, Cave[12] reported a myoma of the jejunum.

Finney,[21] in 1933, reported a myoma of the ileum showing cystic degeneration with hemorrhage. Dixon and Steward,[22] in 1933, reported two cases of leiomyomata of the jejunum, one of which showed myxomatous degeneration.

Rankin and Newell,[13] in 1933, reported from the records of the Mayo Clinic six cases of pure myomata of the small intestine and two of fibromyomata, distributed as follows: duodenum four, jejunum three, and small intestine (not designated) one.

Of the forty-seven cases of myomata of the small intestine recorded in the literature, including the authors' case, twenty-seven were in males and fourteen in females. The sex was not stated in six instances. The ages of the patients ranged from fifteen to seventy-nine years. The tumors were distributed in the intestinal tract in the following manner: duodenum nine, jejunum fifteen, ileum nineteen, and small intestine (not designated) four. In only one case was there any mention made of multiplicity of the tumor. In twenty-four cases the tumors were extraluminar in type, and in twenty-one instances they were intraluminar. The type was not stated in two cases.

REPORT OF A CASE

W. W., a negro man, aged thirty-eight years, was admitted to the surgical service of the Memphis General Hospital May 18, 1931, with a history and the physical findings of intestinal obstruction of four days' duration. The past history revealed severe constipation over a period of three years with an occasional bloody stool.

An exploratory laparotomy, done on the date of admission, revealed a large kidney-shaped mass attached to the external surface of the ileum about 1 m., 83 cm. above the ileocecal junction, situated deep in the pelvis and so twisted on its pedicle as to obstruct the lumen of the ileum. The small intestine above this point was distended and protruded through the opening in the abdominal wall. The gut below this area was collapsed. The mass was removed by dividing the intestinal tract above and below the tumor.

The patient expired before the operation was completed and no autopsy permit was obtained.

Pathological Report.—The specimen consisted of a kidney-shaped mass (*Fig. 3*) weighing 250 gm., measuring 10 x 7 x 6 cm. and bulging the external surface of a portion of the ileum, which was dilated above and collapsed below. The mass was quite firm and covered with a thick hyaline serosa. On section the surface was pink, firm, and composed of coarse, interlacing bundles of tissue. The mucosa of the ileum was ul-

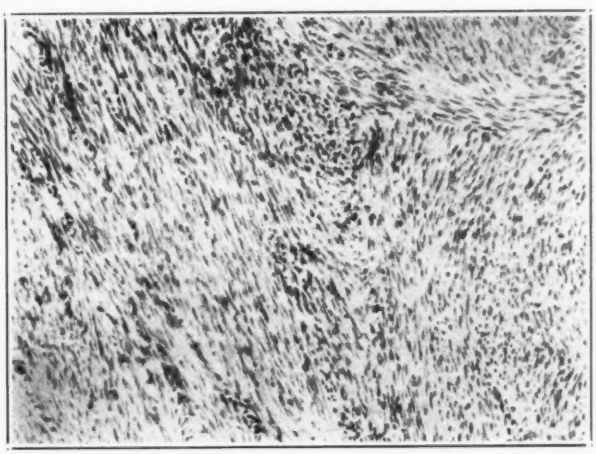

Fig. 4
Myoma of ileum. Note the smooth muscle cells. Photomicrograph x 200.

Fig. 5

Multiple fibromata of ileum. Tumor No. 1 showing the anterior, external surface. Lower end of partly collapsed ascending colon, above; dilated ileum, below. Photograph.

cerated for a square centimeter over the tumor, though the latter was entirely outside the lumen, between the submucous and serous coats.

Microscopic Examination.—The mass was found to be composed of spindle-shaped, smooth-muscle cells arranged in interlacing strands continuous with the muscular coat of the intestine (*Fig. 4*).

Anatomical Diagnosis.—Myoma of the ileum.

In this case of myoma of the ileum, the only clinical symptoms were chronic constipation and intestinal hemorrhage terminating in intestinal obstruction.

MULTIPLE FIBROMATA OF THE ILEUM

Fibromata constitute that group of benign intestinal tumors over which there is the greatest diversity of opinion as to which variations may be included under this heading. It is generally conceded that the true fibroma is of rare occurrence. A brief review of the literature reveals forty-one such cases occurring in the small intestine.

King,[3] in 1917, summarized eight cases of fibromata of the small intestine previously reported and reported a case of his own. In addition he discussed three cases of myxofibromata. After his paper had gone to press, King found two additional case reports of fibromata, one by Means and Forman[23] and the other by James and Sappington,[24] each occurring in the small intestine and giving rise to intussusception.

Clifton and Landry,[7] in 1927, studied all cases of fibromata previously recorded in the literature and reported one case of their own, a fibroma of the ileum. They omitted the case of James and Sappington, which is included in our series.

At the time the paper of Clifton and Landry was published there were twenty-nine cases of fibromata of the small intestine recorded in the literature. These included only authentic reports of fibromata, myxofibromata, fibromyxomata, and myxomata. One case of fibrolipoma and another of adenofibroma were included because the tumor in each instance was composed mainly of fibrous tissue. Clifton and Landry included fibromata, myxomata and variations between these two tumors under one heading because they were considered to have a like primary-cell derivation.

Raiford,[10] in 1932, summarized two fibromata of the duodenum and two of the jejunum from records of the Johns Hopkins Hospital. In the same year, Cave[12] reported a case of fibroma of the ileum, and Larson[25] reported a submucous myxofibroma of the ileum.

Fiedler,[11] in 1932, briefly summarized the literature regarding the clinical aspect of benign tumors of the small intestine and reported a fibroma of the ileum.

Rankin and Newell,[13] in 1933, reported four fibro-

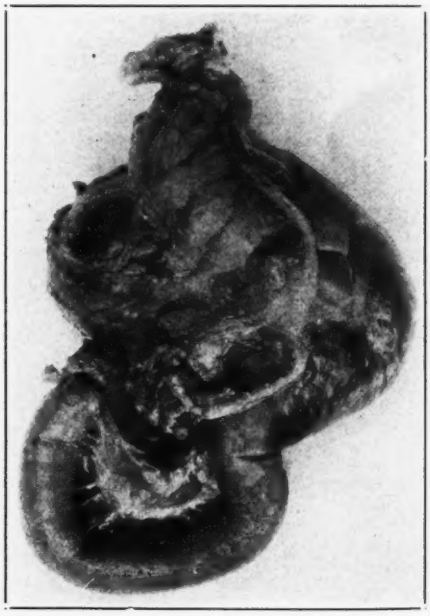

Fig. 6

Multiple fibromata of ileum. Tumor No. 1 showing the posterior, external surface. Partly collapsed cecum with appendix, and end of ascending colon, above; dilated ileum, below. Photograph.

Fig. 7

Multiple fibromata of ileum. Tumor No. 1. Right half of specimen as shown in Fig. 6, on section. Tumor of delicate white fiber is in subserous coat of ileum compressing its lumen, causing obstruction with dilatation of ileum, below, and partial collapse of cecum and ascending colon, above. Photograph.

mata from the records of the Mayo Clinic. Histologically only one was a pure fibroma. The others had undergone myxomatous change or had become hyalinized or calcified to such an extent that it was impossible to determine whether the histogenesis was that of fibroma or myoma. These cases have, however, been included in the present series as fibromata.

Of the forty-two cases reported in the literature, including the authors' case, twenty-one were in females and nineteen in males. The sex was not stated in two cases. The ages of the patients varied from eight to sixty-six years. The tumors were distributed in the intestinal tract as follows: duodenum three, jejunum six, ileum twenty-three, and small intestine (not designated) ten. In thirty-three instances the tumor was intraluminar in type, in eight cases it was extraluminar, and in one case the type was not stated.

In only three cases was there any statement of multiplicity of the tumor, one reported by Duckworth, quoted by King, one reported by Raiford, and the authors' case.

REPORT OF CASE

W. A. C., a negro woman, aged thirty-five years, was admitted to the gynecological service of the Memphis General Hospital May 7, 1931, complaining of a hard mass in the lower right abdomen, first noticed eight months previously. The past history was negative except for constipation. The preoperative diagnosis was fibroma of the uterus.

Laparotomy revealed a large tumor arising from the ileocecal region which was so intimate a part of the external intestinal wall that a portion of the large bowel and ileum were resected during its removal. A second smaller tumor was found in the wall of the lower ileum, and 6 cm. of the ileum on each side of the tumor were resected during its removal. A small subserous fibroid was found and left in the fundus of the uterus. The postoperative course was uneventful.

Pathological Report.—The larger of the specimens (*Figs. 5* and *6*) was composed of 30 cm. of terminal ileum, the cecum with appendix and 8 cm. of ascending colon, of which the tumor, weighing 1,140 gm. and measuring 16 x 13 x 8 cm., was a part. The tumor was completely covered with peritoneum. On section (*Fig. 7*) it was found to be outside of the muscular coat of the ileum, not protruding into the lumen, but compressing it and having caused obstruction with dilatation of the ileum above and some collapse of the cecum below. The surface was relatively avascular and composed of interlacing whorls of firm, white, connective tissue. The smaller specimen (*Fig. 8*) consisted of 18 cm. of ileum containing a tumor weighing 80 gm. and

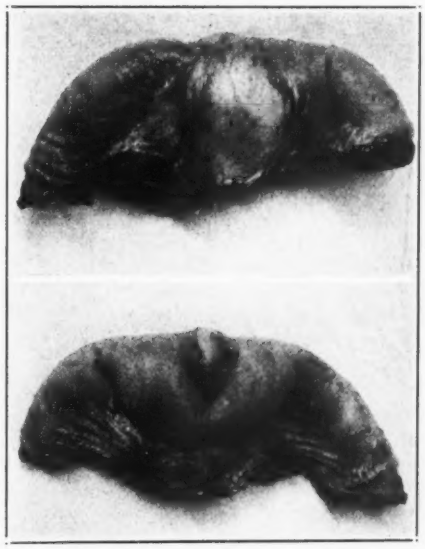

Fig. 8

Multiple fibromata of ileum. Tumor No. 2. External surfaces. Dilated ileum above tumor; collapsed, below. Photograph.

Fig. 9

Multiple fibromata of ileum. Tumor No. 2. Posterior half of specimen as shown in lower photograph of Fig. 8. The white, fibrous tumor takes origin in subserous coat and projects into lumen, causing obstruction with dilatation of ileum above and collapse, below. Photograph.

measuring 5 x 3 x 3 cm. It was similar to the larger one except that on section (*Fig. 9*) it protruded into the lumen and caused obstruction with dilatation of the ileum above and collapse below.

Microscopic Examination.—The tumors revealed interlacing strands and bundles of fibrous tissue (*Fig. 10*) arising from the subserous coat of the ileum.

Anatomical Diagnosis.—Multiple fibromata of the ileum.

In this case of multiple fibromata of the ileum, the only clinical symptoms referable to the condition were an abdominal mass and chronic constipation.

SUMMARY AND CONCLUSION

A brief review of the literature regarding benign tumors of the small intestine reveals twenty-five cases of hemangiomata, forty-seven of myomata, and forty-two of fibromata. These tumors were distributed as follows: duodenum eighteen, jejunum twenty-six, ileum forty-six, small intestine (not designated) twenty-two, and the entire intestine two (*Table 1*).

Table 1

FREQUENCY OF OCCURRENCE AND LOCATION

	Hemangioma	Myoma	Fibroma	Totals
Duodenum	6	9	3	18
Jejunum	5	15	6	26
Ileum	4	19	23	46
Small intestine	8	4	10	22
Entire intestine	2	0	0	2
Totals	25	47	42	114

The ages of the patients varied from two months to seventy-nine years. The tumors showed a slight tendency to occur more frequently in males than in females. By far the greater number of the tumors were single rather than multiple; and tended to be intraluminar in type (*Table 2*).

Table 2

MISCELLANEOUS FINDINGS

	Hemangioma	Myoma	Fibroma	Totals
Age Variation	2 Months to 79 Years	15 to 79 Years	8 to 66 Years	
Sex				
Males	10	27	19	56
Females	13	14	21	48
Not stated	2	6	2	10
Totals	25	47	42	114
Multiple	9	1	3	13
Single	16	46	39	101
Totals	25	47	42	114
Intraluminar	23	21	33	77
Extraluminar	2	24	8	34
Type not stated	0	2	1	3
Totals	25	47	42	114

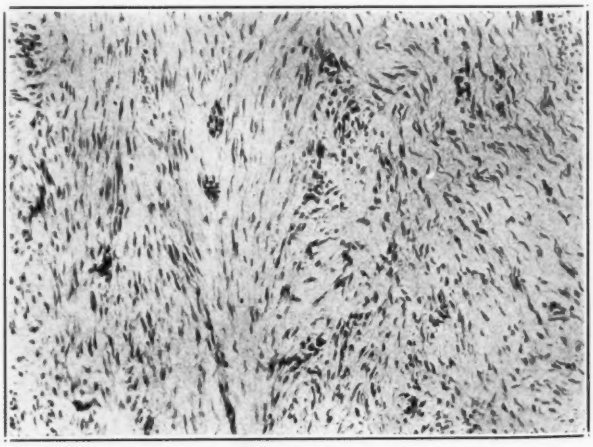

Fig. 10

Multiple fibromata of ileum. Tumor No. 1, showing delicate, wavy fibrous tissue. Tumors Nos. 1 and 2 show the same structure. Photomicrograph x 200.

The most common clinical symptoms of these tumors were (a) intussusception; (b) obscure intestinal disorders; and (c) anemia.

The following three illustrative cases have been reported: (a) cavernous hemangioma of the duodenum, (b) myoma of the ileum, and (c) multiple fibromata of the ileum.*

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DISCUSSION (*Abstract*)

Dr. Sim Hulsey, Fort Worth, Tex.—These cases bring to mind a case of my experience of another type of benign intestinal tumor with resulting death. The patient was a woman upon whom an operation had been done two years previously for intestinal obstruction and the ileum telescoping into the cecum was relieved. On this latter occasion of her illness there were again the symptoms of obstruction. Apparently operation was permitted too late and the patient died. At autopsy many submucous lipomas were found in the lower one-half of the ileum. These were soft, yellowish white in color, pendulous and their diameters varied from 0.5 to 5 cm. One of the larger ones had been caught and carried downward, with intestinal obstruction due to intussusception. Lipomas of the intestinal tract have been mentioned in the literature, but comparatively rarely. They are, of course, benign, but can be considered malignant from position. This case was the first of intestinal lipomatosis in my experience.

*We are indebted for the photographs and photomicrographs of these cases to Mr. Joseph L. Scianni, Artist, University of Tennessee, Pathological Institute.

ABSCESS OF THE LIVER COMPLICATED BY DUODENAL FISTULA*

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and

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This paper is limited to a brief discussion of liver abscess, of duodenal fistula and of the treatment used in aiding the recovery of a patient having both conditions.

Abscess of the liver is most often a complication of *Entameba histolytica* infection. It may also follow suppuration of abdominal organs, especially when the venous return is through the liver. A few cases come from liver trauma. It is limited largely to males. Matas[1] thinks that 80 per cent of the cases of solitary liver abscess are due to amebic infection of the colon. Gessner,[2] in a series of ninety-six cases, found that fifty-six were amebic with 33.9 per cent mortality; and forty were pyogenic in origin with 37.9 per cent mortality. Boland[3] reported fourteen cases of amebic liver abscess, with 35.7 per cent mortality, and five pyogenic cases with 60 per cent mortality. Herrick[4] reported forty-seven cases, probably all amebic, with 23.4 per cent mortality.

Matas found an incidence of seven per ten thousand white hospital population in New Orleans between 1914 and 1924. Probably this is becoming less with better treatment of amebiasis and earlier diagnosis of abscess by x-ray demonstration of the characteristic elevation of the right diaphragm.

A large tender liver with emaciation, fever and leucocytosis is typical. History of diarrhea is not essential. Amebas were found in the stools in only one of Boland's fourteen cases of amebic abscess. Even after the abscess has been identified and drained, difficulty may be expe-

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rienced in etiological classification. In our experience, amebas have not been found in the pus, which is most often reported without organisms. Even if pyogenic organisms are present they may be from a secondary infection.

During the past five years we have operated upon six cases of liver abscess, all in white men. Five were considered amebic in origin and one was traumatic. Two died after operation, giving a mortality of 33 per cent, which approximates the results of others. It may be surmised that liver abscess is not rare in South Carolina. Adequate treatment of amebic cases should lessen the incidence of abscess. When diagnosis is in doubt, laparotomy is advocated rather than needling. This should not be delayed, for the condition is progressive and the outcome may be jeopardized.

Duodenal fistula occurs even less frequently than liver abscess. Kittelson[5] reports a total of ninety-six cases in the literature since 1865, with surgical mortality of 50 per cent and nonsurgical of 27.7 per cent. Willis and Mora[6] report one case, and Baker[12] two additional cases, making a total of ninety-nine to date.

Death is caused by starvation, dehydration and toxemia from loss of digestive fluids. It is estimated by Elliott[7] that sodium accounts for 90 per cent of the total base of blood serum and extra-cellular fluids. The other bases, calcium and potassium, are of less importance in maintaining the normal acid-base balance. Hence, in supplying fluids and salts to replace the large amounts lost through the fistula, normal saline solution is ordinarily sufficient. The problem in maintaining normal fluid and mineral equilibrium may become more complex in severe cases and sodium chloride alone may be inadequate. The buffer salt solutions, as emphasized by Hartmann,[8] better maintain the normal salt balance and are indicated in prolonged cases with depletion.

The occurrence of duodenal fistula is associated most frequently with trauma from operation in the upper abdomen. Kittelson's[5] analysis shows the incidence after gallbladder operations to be 31 per cent; after perforated duodenal ulcer, 23 per cent; after nephrectomy and resection of the stomach, each 0.8 per cent; and after various other abdominal lesions, 33 per cent. He found that fistulous openings distal to the papilla of Vater are more destructive and have a higher mortality. The prognosis after cholecystectomy is better than after nephrectomy. The continuous loss of pancreatic fluid is fatal (Walters and Bollman[9]).

The diagnosis of external duodenal fistula is obvious. Fluids given by mouth escape through the fistula into dressings. In a short while excoriation of the skin from partial digestion by trypsin and intestinal secretions which pour over it takes place. Starvation, dehydration, demineralization and local irritation combine to cause progressive emaciation and weakness, dependent in degree upon how much of the food intake escapes through the fistula and how much is retained.

The operative closure of the opening in the duodenum has such a high primary mortality as to be practically prohibitive. Consequently, the endeavor is made to provide adequate alimentation until the fistula can close spontaneously. Attempts at control by packing, or plugging, have been uniformly disappointing, although Baker[12] has recently reported two cases successfully treated by injections of Beck's bismuth paste. Einhorn's[11] plan of feeding through a duodenal tube passed by mouth is, in our opinion, ideal if the pylorus is patent and if the distal end of the tube can be passed beyond the opening of the fistula. Willis and Mora recently succeeded in passing a soft rubber catheter directly through the fistulous tract into the duodenum and fed the patient for two weeks until the fistula had practically healed. If this cannot be accomplished, feeding by jejunostomy as advocated by Stuart McGuire[10] may be the only way to preserve life. Attempts at prevention of tryptic digestion of the tissues by continuous drip of peptone or albumin into the wound are as unsatisfactory as is a weak hydrochloric acid drip. Potter[13] advocates postural drainage on a Bradford frame to prevent skin excoriation.

CASE REPORT

A white man (J. J. J.), aged 52, a blacksmith and farmer, was admitted to the South Carolina Baptist Hospital October 4, 1932, complaining of fever, general weakness and swelling in the right upper abdomen. His illness began four months before with a depleting diarrhea. The stools contained mucus and pus. For the first six weeks he was confined to bed and his weight fell from 180 to 115 pounds. Fever developed with loss of appetite, nausea and vomiting. In the preceding month a diffuse swelling had appeared in the right upper abdomen.

The past history was that of a hard-working out-of-doors man, who had had no illnesses except mild attacks of diarrhea each spring for several years, and the passage of urinary gravel three and six years before.

Physical examination revealed the mere skeleton of a large-framed man, very weak, lying passively in bed. The skin was of a pale and putty-like tint. Pyorrhea was marked. The lungs and heart were negative. The blood pressure was 90 over 60. There was an indefi-

Fig. 1
Preoperative. X-ray showing enlarged liver, elevated diaphragm and depressed right kidney.

nite, slightly tender mass in the right upper quadrant of the abdomen without muscle spasm or rigidity. The spleen was not enlarged. The temperature was 100°, pulse 90, respiration 23.

Laboratory examinations showed the urine to be negative. White blood cells were 4,000, with polymorphonuclears 74 per cent and hemoglobin 60 per cent. Repeated stool examinations during convalescence were negative for ameba.

X-ray and fluoroscopic examination (*Fig. 1*) October 4 showed a much enlarged smooth liver shadow with displacement of the right kidney downward. The domes of the diaphragm were equally elevated. The left was more mobile than the right.

The morning after admission, under spinal anesthesia, the abdomen was explored. An inflammatory mass was found filling the right upper quadrant from a ruptured massive solitary abscess of the right lobe of the liver, which had been localized by the pylorus, duodenum, transverse colon and omentum. More than a quart of typical liver pus without odor, thick and pink in color was evacuated and the abscess cavity in the liver was packed with gauze to control bleeding. Dependent drainage was secured through a stab wound in the flank below the right costal margin. No organisms were found in the cultured pus.

The following day, after the patient took water, the dressings became wet. Methylene blue solution taken by mouth soon stained the dressings, proving a communication between the upper alimentary tract and the abscess cavity. Food particles later appeared in the drainage wound. X-ray examination (*Figs. 2* and *3*) on the ninth postoperative day, after bismuth administration, showed a fistulous tract from the first portion of the duodenum to the exterior, communicating with the abscess cavity in the liver. The case was further complicated by a profuse hemorrhage from the stab wound on the twelfth postoperative day, which was controlled by repacking and by blood transfusion.

In spite of several blood transfusions and dai'y hypodermoclysis of glucose in normal saline solution, dehydration and toxemia progressed and recovery was almost despaired of. On the twenty-seventh day a Rhefus duodenal tube was passed by mouth several inches beyond the fistulous opening of the duodenum (*Fig. 4*). Methylene blue so'ution introduced into the tube did not show in the wound. Warm duodenal feedings of high caloric value totaling 2,500 calories per day, but not exceeding 240 c. c. per feeding, were given through the tube every three hours and normal salt solution

Figs. 2 (top) and 3 (bottom)
(Retouched) Ninth postoperative day showing abscess cavity with fistulous tract from duodenum.

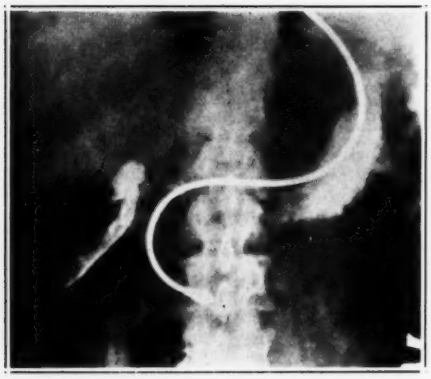

Fig. 4
(Reconstructed) Showing duodenal tube in place beyond fistulous opening.

and water dripped in at intervals day and night. No food was given by mouth for the forty-five days that the tube was in place and liquids were discouraged in order to keep the stomach at rest and to allay peristalsis. In addition to this, physiological buffer salts (Hartmann's formula) were given daily in glucose solution by hypodermoclysis throughout the critical period because dehydration and toxemia were not being controlled. The patient was placed on the left side during and for half an hour after feeding. This, combined with wound packing, checked drainage and lessened skin excoriation.

Improvement was soon apparent; fever gradually subsided from 102° to normal by the seventeenth day. Diarrhea throughout was troublesome, and no doubt was aggravated by the tube slipping too far into the jejunum, and by overfeeding. The position of the tube was checked by the fluoroscope and a measured length was left outside, which was of value in replacing the tube when it became plugged. Emetin was used to combat his amebic infection. Arsenicals were avoided on account of the damaged liver.

The surgery performed in this case was relatively simple. Postoperative care was more difficult. The patient was discharged, ambulatory, on the seventy-ninth day.

On October 20, 1933, examination showed that he had regained normal strength and was within five pounds of normal weight. He had worked at hard farm labor all summer and felt well. Fluoroscopic examination showed a normal stomach, duodenum and liver.

Experience with previous patients with liver abscess who died of toxemia and disturbance of water balance demonstrated the necessity of sufficient fluids, salts and nutrition. The fatal cases received normal saline and water in large amounts, but evidently the deficit was never overcome. More liquids and salts than seem possibly necessary must be given. They are life-saving.

CONCLUSIONS

A case has been reported of liver abscess with the rare complication of duodenal fistula. The postoperative use of the duodenal tube passed by mouth beyond the fistulous opening to insure adequate jejunal alimentation, combined with hypodermoclysis of normal salt solution and physiological buffer salts in large amounts, were the measures that contributed to recovery. The more general employment of this regime is suggested for cases of duodenal fistula when the pylorus is not obstructed. So far as we know, this is the third case treated in this way and is the only reported case of liver abscess complicated by duodenal fistula.

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DISCUSSION (*Abstract*)

Dr. Frank K. Boland, Atlanta, Ga.—Among twenty-two cases of abscess of the liver reported from the Emory University Division of the Grady Hospital, Atlanta, there were no instances of duodenal fistula or fistula into other viscera. In 563 cases of liver abscess collected in 1887 by Cyr ("*Traité pratique des makadies du foie*"), and recorded in Rolleston and McNee's work on "Diseases of the Liver, Gallbladder and Bile Ducts," eighty-three cases, or 15 per cent, were operated upon, and rupture occurred in 159, or 28 per cent. It should be remembered, however, that interesting examples of perforated hepatic abscess are more apt to be put on record than more commonplace cases. Since in abscess of the liver the enlargement takes an upward direction, rupture is more liable to take place through the diaphragm into the lung, pleura and pericardium than into the peritoneum and abdominal organs. Fifty-seven per cent of Cyr's cases perforated through the diaphragm. Only thirteen cases perforated into the intestine, and most of these were into the duodenum.

So Dr. Bunch is reporting a rare complication of

hepatic abscess, and I wish to congratulate him upon the logical and ingenious treatment he used and the excellent results which he obtained.

Amebic disease seems to be on the increase in this country, and as Dr. Bunch observes, more care should be given to the early diagnosis and treatment of the condition so as to prevent such serious complications as liver abscess. Physicians should be on the lookout for amebic dysentery in every community. The stools of all patients with dysentery ought to be examined for amebae. The plasmodium seems to be more easily discovered in stools in cases without complications than after complications have taken place. It has already been stated that neither the ameba nor its encysted form is often found in stools in cases of hepatic abscess, nor is it found in the pus from the abscess, facts which sometimes delay correct diagnosis of abscess of the liver. In a case of amebic ulceration of the colon which I saw recently, no amebae could be demonstrated in the patient's stoo's, but autopsy revealed ulcers swarming with amebae. The ameba appears to be loth to leave its comfortable resting place in an ulcer or in the wall of an abscess cavity, where it is as snug as a bug in a rug. It can be pronounced as an axiom that the presence of numerous amebae in a stool is strong evidence against the existence of the complications of amebic dysentery.

One slide of amebic abscess of the liver may show how the hepatic enlargement extends upward, while another slide of carcinoma of the liver may show how the enlargement is downward into the peritoneal cavity. Another slide demonstrates the *Endameba histolytica* enclosing red blood corpuscles, which the commoner *Ameba coli* seldom does.

Dr. J. Russell Verbrycke, Jr., Washington, D. C.—It is not likely that any of us will ever see another case of the unusual combination of conditions reported by Dr. Bunch. But anyone can, at any time, be confronted with that terrible complication of duodenal fistula, than which there is nothing much more to be feared by the surgeon.

When Dr. Bunch speaks of "skin excoriation" he puts it mildly. The condition is one of tissue destruction, so rapidly progressing that it is almost as if an auger were reaming out the hole. In 24 hours, from a tiny opening the erosion may spread to a size of a half inch, and grow larger by the hour. Death is by no means entirely, or perhaps even primarily, due to loss of food and body fluids.

In discussing his treatment the essayist did not mention what, in my experience, has been by all odds the most satisfactory method. Its utilization does not preclude the simultaneous use of the duodenal tube. It consists of the introduction of a tube as far down the fistula as it can be inserted, followed by continuous suction by means of the same small electric apparatus that the anesthetists use in tonsil cases. The suction must be started at the earliest possible moment and cannot be discontinued as long as there is any return. The size of the tube is progressively diminished as the fistula closes in.

Early recognition of a fistula before tissue destruction has made much headway is very important and can readily be accomplished by having the patient drink methylene blue colored water and noting whether the blue color appears on the surface in a few minutes.

FRACTURES OF THE OS CALCIS AND ASTRAGALUS*

By WRIGHT CLARKSON, M.D.,
and
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Fractures of the astragalus rarely occur alone. Isolated injuries are occasionally reported, usually the result of direct violence, but the large majority of these fractures are associated with fractures of the calcaneus. Since this is true, and because the same principles of diagnosis and treatment apply as well to one as to the other, we shall limit this paper to a discussion of injuries of the os calcis.

Fractures of the os calcis comprise approximately 2 per cent of all fractures, and of this number 90 per cent are the compression type. The frequency of their occurrence, the large number of men who are incapacitated following haphazard treatment, and the fact that these injuries are nearly always sustained by those whose economic status is almost wholly dependent on the proper function of their feet, make careful treatment of them most important. The hazards of industry have increased as machinery has been added with the consequence that it is not unusual to see a fracture of almost any portion of the osseous system. However, there is no fracture, we believe, which so frequently requires an open operation to obtain a satisfactory result as do fractures of the calcaneus. This fact alone places these injuries among the most hazardous type as regards future impairment. Fortunately for these patients, the recent trend in their treatment has for its object the close approximation of fragments and the correct apposition of the astragalocalcaneal joint.

Practically all fractures of the os calcis are the result of a fall with the patient landing squarely on his feet. We occasionally see fractures involving the mesial and lateral processes of the tuber calcanei. Although these require treatment which will reduce the fragments, they are not so serious as the usual compression type because the subastragaloid joint is not frequently disturbed. In the diagnosis of these fractures it is always wise to remember that the clinical manifestations may be very mild in con-

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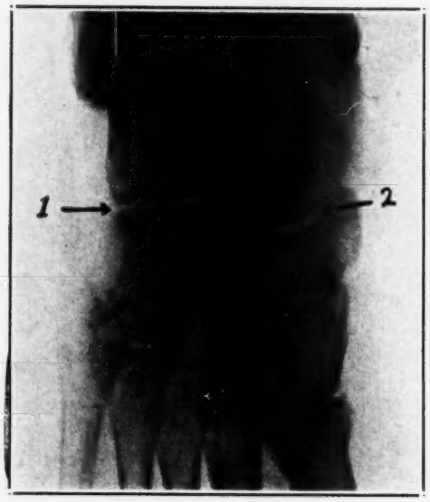

Fig. 1
Usual anteroposterior view showing the anterior aspect of the os calcis. (1) Calcaneocuboid joint, and (2) astragaloscaphoid joint.

trast to the severity of injury. Keeping this fact foremost in our mind, we should consider that every patient who has fallen on his feet from a considerable height has a fractured os calcis until otherwise proven by careful roentgen studies. Such care in the handling of these cases would have prevented many of the disabling deformities which we have seen after it was too late for them to be corrected.

In fractures of the os calcis, as in fractures of other bones, there are all degrees of injury, but in the great majority of those which result from a fall, the posterior fragment of the bone is displaced upward, causing a flattening of the longitudinal arch. The bone is compressed so that the lateral portion is displaced outward and impinges against the external malleolus. Almost invariably the subastragaloid joint is disturbed and frequently the calcaneocuboid. Any one of these conditions necessitates correction and not the mere application of a plaster cast. One can readily picture the disabling deformity which will ensue should the bone be allowed to heal without approximation of the fragments, or correction of the deformed joints.

In the diagnosis of these injuries an accurate history is extremely important. Almost invariably patients with fractures of the os calcis will give a history of having fallen from a height of five feet or more. Not all patients who have met such an accident will have a fracture of this bone, but if we fail to make roentgenograms of these cases when they complain of pain, however mild, we will deprive many of their only opportunity for proper treatment. We will also subject ourselves to criticism when later, pain will send a certain number of these cases to another physician who will discover the true nature of their injury after it is too late to obtain a satisfactory result.

Carefully made roentgenograms are indispensable. We want to stress the necessity for an adequate number of films made at various angles. The surgeon must know the extent of the injury to be able to accomplish a satisfactory reduction. Too often treatment is inadequate because roentgen studies are incomplete. The usual antero-posterior and lateral views are not sufficient. These alone will not disclose the extent of injury nor the position of the frag-

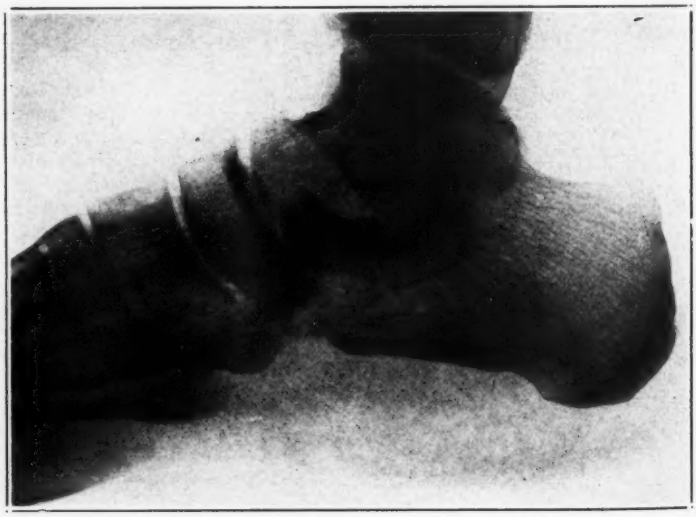

Fig. 2
Usual lateral view of the os calcis.

Fig. 3
Arrow denotes the astragalocalcaneal joint. This joint is usually well outlined in the lateral view taken with the heel slightly elevated.

ments, and may, therefore, lead one to believe that the application of a plaster cast is all the treatment necessary. In addition to the anteroposterior and lateral views, we always take one film through the posterior aspect of the foot. We believe this can be done with less difficulty and more comfort to the patient if we have him lie prone on the table with the cassette against the sole of the foot and the tube pointing downward toward the feet angulated about forty-five degrees from the horizontal. An adequate interpretation of these roentgenograms must clearly portray to the surgeon the position of the fragments, whether there is upward or lateral displacement, the joints involved, and most important of all, he should know the degree of injury to the subastragaloid joint.

In the treatment of this injury as in the treatment of many other conditions the best results can be obtained only by close cooperation between surgeon and roentgenologist. Both must be familiar with the mechanics involved and the possibilities of deformity.

It is not our intention to discuss the treatment of these injuries other than to say that too often treatment consists only in fixation or correction of the deformity and wholly neglects the astragalocalcaneal joint. Such methods will inevitably result in disabling pain in a high percentage of these patients.

A guarded prognosis should be given in all cases of fractures of the os calcis, as the injury will quite often result in a painful foot no matter what method of treatment is used. It is important to remember, however, that disability is practically always much greater in those patients who have been treated only by immobilization, no attempt having been made to correct the deformity of either the bone or subastragaloid joint. Such patients are flat-footed, are unable to walk on uneven surfaces without pain, and find it difficult to rise on their toes because of a loss of leverage due to the shortening of the os calcis. The heel is abnormally wide and frequently displaced outward because the compressed bone has not been restored to its normal contour. Therefore, every step causes the lateral aspect of the os calcis to impinge against the external malleolus. With this there is added strain on an already traumatized subastragaloid joint in which there has developed a traumatic arthritis, which, in most cases, is the primary cause of persisting pain and disability.

Fig. 4
Projection of the os calcis with the rays directed through the posterior aspect of the foot as illustrated in Fig. 5.

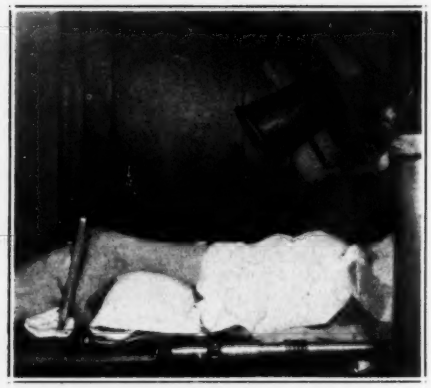

Fig. 5

Illustrating the relative positions of x-ray tube and patient's foot in obtaining the roentgenogram shown in Fig. 4.

Bone irregularities and exostoses frequently appear on the under surface of the bone. These project into the soft tissues and become exceedingly painful when the patient attempts to walk. Such findings seldom occur in those fractures where close approximation of the fragments is obtained, as there is very little secondary callus, union being direct. Patients with any one of the deformities just described frequently find it impossible to return to their original occupation, and often become an economic burden to their family or community. The possibility of such an unfortunate climax to what may occasionally seem a trivial injury should cause us all to exercise extreme care and judgment in the diagnosis and treatment of any suspected fracture about the heel. As mentioned previously, a certain number of these injuries will result in a painful foot, no matter what form of treatment is used. When pain does occur in those cases where the fragments were approximated and the mechanics of the foot restored, it is almost certainly the result of a traumatic arthritis in the astragalocalcaneal or calcaneocuboid joint, and relatively simple operations have been devised for the relief of this pain.

The problem of fixing responsibility and compensation disputes frequently arise as a consequence of these injuries. In all such cases a careful history of any previous injury is extremely important. It not infrequently happens that a man employed by a company receives a trivial injury to which he attributes all of his disability and on careful questioning we find that this is the result of an injury received months or years prior to the time he started work with the company in question. It is true that the recent injury may cause an exacerbation of his symptoms, yet it is not primarily the causative factor and therefore the company should not be made liable. Only recently such a case came to our attention.

The patient was referred for examination of the left foot by a railway surgeon. He was complaining of pain and some swelling, the result of a minor accident five days before. He insisted that all his disability was the result of this injury and, therefore, placed all responsibility on the railroad. However, roentgen examination showed an old healed fracture of the os calcis with the posterior fragment displaced upward, a flattening of the longitudinal arch, a compression of the superior aspect of the bone and a traumatic arthritis of the astragalocalcaneal joint. There was no evidence of recent bone injury. On careful questioning and after he was told that at some time in the past he had fallen a considerable distance and landed squarely on his feet he admitted that seven years before he had

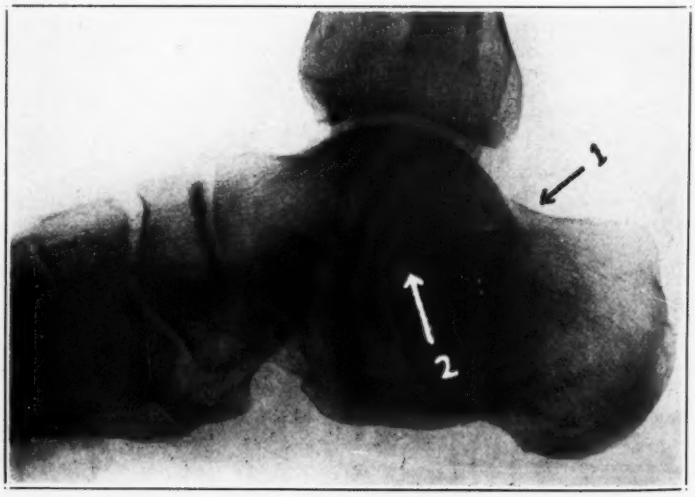

Fig. 6

Old compression fracture of the os calcis of the usual type resulting from a fall. (1) Fracture of the superior aspect of the calcaneus. (2) Traumatic arthritis of the astragalocalcaneal joint with almost complete obliteration of the joint space.

received such an injury from a height of fifteen feet. He also revealed that at this time he was treated three weeks for "sprained heels," but no roentgen examination was made. During the interval between the old and recent injury he had at times suffered with pain in both feet when walking. Because of this history we examined the right foot also and found an old healed fracture of the right os calcis. The report rendered in this case was as follows: 'Old healed fractures of both os calces with considerable deformity of the left foot and a traumatic arthritis of the left astragalocalcaneal joint. The patient's symptoms we believe are the result of this arthritis. We can find no evidence of recent bone injury."

This case not only illustrates the importance of the history in fixing responsibility, but emphasizes the necessity of making roentgenograms of every patient who falls on his feet and complains of pain, however trivial it may be.

CONCLUSIONS

(1) Fractures of the os calcis occur frequently and usually are the result of a fall.

(2) Any patient who has fallen a distance of five feet or more, landing on his feet, should be studied with the possibility of a fractured calcaneus in mind.

(3) Roentgen studies should include several views and the astragalocalcaneal joint should be clearly defined on the roentgenograms.

(4) Treatment too often consists of immobilization and neglects the deformity of the bone and subastragaloid joint.

(5) The prognosis should be guarded in every case, even with proper treatment, as pain frequently persists, the result of a subastragaloid traumatic arthritis.

(6) The history, when correlated with the roentgen findings, is frequently very important in establishing responsibility.

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Medical Arts Building

DISCUSSION (*Abstract*)

Dr. S. O. Black, Spartanburg, S. C.—Dislocations of the astragalus are of two main varieties, incomplete and complete. In the former, the os calcis and scaphoid are displaced from their normal relations with the inferior and anterior articular surfaces of the astragalus. In the latter, the astragalus is rotated and dislocated at its articulations with both os calcis and with the tibia. These injuries result from severe violence and in the incomplete type, the foot is displaced outward or inward, the head of the astragalus lying to the medial or lateral side of the scaphoid as the case may be.

Recent cases can occasionally be reduced by closed manipulation, whereas in older and more extensive injuries it will often be necessary to resort to sugery; the procedure being to replace with or without arthrodesis or excision of the astragalus *in toto*.

Fractures of the astragalus occur in 0.004 per cent of all cases of skeletal fractures and dislocations, according to Massachusetts General Hospital statistics. There are four general types. The transverse fracture through the body or neck is usually associated with complete dislocations of the posterior fragment either backward or inward. Often the circulation to this fragment is cut off and the procedure of choice is to do an astragalectomy with displacement of the foot.

Os calcis fractures are by far the most frequent of all tarsal injuries. In a series of 4,536 cases at the Massachusetts General Hospital they were 38, or over 1 per cent of the total They are peculiar to adult life and most frequent between forty and fifty years of age. They are usually due to falling or landing on the rigid heel instead of the flexible forefoot.

Fractures of the body of the bone are of two general classes: crushing fractures and fissure fractures. The former are due to falling for a considerable distance and are accompanied by extreme comminution of the bone with flattening and spreading. The fissure fractures have much less comminution and spreading; the main fissures usually take the form of the letter V.

Severe fractures of the os calcis are notoriously disabling. The period of recovery has been estimated at from four to twelve months or longer. All types of effort have been made to reduce and replace the fragments into a form and shape resembling normal. Most of these have been unsuccessful with resulting pain, tenderness, deformity and inability to stand for any time or to walk for any distance. Wilson, of Boston, resorts to subastragalar fusion wherever the fracture runs into the subastragalar joint. This operation, though difficult to perform, is followed by less disability than any other procedure he has tried in severe fractures of this bone. It lessens the pain, corrects the existing valgus position and permits the removal of bone spurs or sharp edges if any be present. It, of course, prevents lateral motion of the foot, but there is no motion anyway in nine cases out of ten.

In recent cases of os calcis fracture with slight or no bony deformity or in which there is absence of irregularity in the subastragalar joint, the injury should be treated by fixation in plaster after such manipulation as may be necessary.

In the evulsion type of fracture, where the posterior end of the bone is detached or pulled upward by action of the tendo-achilles, the procedure of choice is open operation, to restore the bone to its normal position

and to hold it there either by suture or by means of a bone peg.

Dr. James Thomas Tucker, Richmond, Va.—Dr. Barker has presented us with some beautiful x-rays of fractured os calces. The immediate operative treatment of fractured os calces by subastragalar arthrodesis, as advocated by Dr. Philip Wilson, of Massachusetts General Hospital, under whom I had the pleasure of receiving some of my training, has become obsolete for those who have worked in and seen the results of Dr. Lorenze Böhler's clinic in Vienna. I have recently returned from a four months' stay there and I shall show you some slides of his methods of reduction.

In a normal foot, if a line is extended along the plane of the subastragalar joint backward and another line is drawn along the plane of the tuberosity of the os calcis backward, the angle between these lines would be between 35 and 40 degrees. When the os calcis is fractured, this angle is reduced from 35 to 0 degrees. Böhler's reduction is an effort to bring this "tubo-joint" angle to the normal 35 degrees. By a pin through the os calcis and a 60-pound pull downward and backward, this angle is restored. Another pin is placed three-fingers' breadth above the malleolus in the tibia. A screw compression apparatus is applied to correct the lateral spreading of the os calcis and immediately a non-padded plaster cast is applied incorporating the two pins, which maintains the reduction.

The open subastragalar arthrodesis advocated by Wilson becomes obsolete when one has seen and tried Böhler's method; but to those who do not know the Böhler technic, the Wilson method is the safest and surest way to lessen disability and restore function.

END-RESULTS OF COXA PLANA AS RELATED TO TREATMENT*

By GUY A. CALDWELL, M.D.,
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The treatment of coxa plana has not been given sufficient consideration. Since scattered statistics show that one out of five patients in the cured stage has a real limp associated with restricted motion and some pain, and about half of these have reduced working capacity, it is essential that we determine whether or not treatment helps, and if so, what is the best method. Observations published in 1926-'27 indicate that the optimistic prognosis given by earlier writers is not wholly warranted. While many have written of the etiology, pathology and symptomatology of coxa plana, few have reported end-results and none has analyzed the results in relation to the methods of treatment employed. Such references to treatment as exist in the texts and literature are varied and somewhat conflicting.

I should like to limit this discussion to the progress and end-results of coxa plana following various procedures of treatment and the alteration of these by various orthopedic measures in common use. I shall present a few cases which have been under observation for sufficient time to form a basis for discussion. Their number is obviously too small to warrant general conclusions, but it is hoped that a liberal discussion from those assembled will add sufficient material to enable us to arrive at some conclusion as to the most effective treatment for the active stage of the disease.

The most complete series of end-results that has been published are those by Flemming-Moller[1] and by Legg.[2] The former reported in 1926 seventy-four cases, of which he says: "I have been able to follow the entire course of the disease, so to speak." His analysis is most accurate in regard to persistent symptoms, gait and limitation of movement, but he does not include the duration of the disease, the character of treatment nor the age of the patients when his final observations were made. His report indicates that, when healed, 39.2 per cent have no noticeable changes; 39.2 per cent show very slight dragging of the leg; 9.4 per cent retain a permanent limp with considerable restriction, and 12.2 per cent have continual pain in the hip and restricted working capacity. He believes that the most important factor in prognosis is the age at onset of the disease. He says that in the perfectly healed cases, the average age at the beginning is six years, in those healed with slight restriction, nine years, and in those with poor results, twelve years. His concluding sentence is: "It is thus, in reality, the age of the child that is determining for the extent of final deformity."

Legg, in 1927, analyzed the terminal results of forty cases. His discussion emphasized the two types of the disease commonly seen, namely, the mushroom and cap types, and indicated that the prognosis depends entirely upon the type. He believes that the mushroom type, in general, gives a better result than the cap type. In regard to prognosis and treatment, he says:

"It always may be said that ultimately the cases of both types will never be incapacitated, although the majority will tire after long standing and walking, or after movements that require extreme abduction, such as riding. Slightly more than half the cases will probably show a persistent limp.

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*Read in Section on Bone and Joint Surgery, Southern Medical Association, Twenty-Seventh Annual Meeting, Richmond, Virginia, November 14-17, 1933.

"There is considerable discussion in regard to the appropriate treatment for this affection. It must be admitted that while any process which suggests a weakening in bone structure is going on in the hip joint, it would seem theoretically sound treatment to allow no weight-bearing. It has been my experience, however, that relief from weight-bearing has in no way affected the end-result: that is, in those cases in which I have not allowed weight-bearing, the end-result has not varied in any way from those in which weight-bearing on the affected leg throughout the course of the disease has been allowed."

In the discussion of Legg's paper, Kidner said:

"These hips should be kept free of weight-bearing for a very long period of time. If they are so held long enough, they will show far better results and less shortening than if the patients are allowed to walk."

Ryerson, discussing the same subject, said:

"Plaster-of-paris fixation should not be used. I personally believe that encasement in plaster-of-paris actually produces bone atrophy, and definitely delays healing. Weight-bearing should not be permitted in cases of Legg's disease, because the femoral head is softened and will become flattened if allowed to press against the acetabulum. The proper treatment, in my opinion, is the use of the Thomas caliper splint, continued until ossification is complete as shown by x-ray pictures; some cases require two or three years, and some only a few months. I do not think they should be left untreated, and I do not think they should be treated by plaster-of-paris. I think, as Dr. Kidner does, that they should be treated by relief from weight-bearing until relief is no longer necessary."

In some of the current texts on orthopedics there is considerable variation in emphasis on methods of treatment employed. Jones and Lovett[3] suggest:

"Some restriction of walking, protection from weight-bearing in cases which are hard to control, and, in exceptionally severe cases, fixation of the joint should be temporarily added to protection from weight-bearing."

Whitman[4] outlines a similar plan. Campbell[5] states:

"Good function may result in those receiving no treatment, but recovery with less impairment of function is accomplished when efficient measures are instituted."

Among the measures suggested are: recumbency with extension when the process is acute; manipulation followed by plaster applied with the limb abducted 45 degrees when there is adduction deformity; a plaster spica for three months, followed by a Bradford abduction brace for three to six months longer, or until the roentgenogram demonstrates that the process has subsided.

Those most familiar with the disease thus differ concerning treatment, some holding that all measures of protection are useless; some, that relief from weight-bearing is the only essential, while others hold for a considerable period of fixation in plaster or by traction followed by prolonged support in splints or braces. In view of these wide differences of opinion, I should like to present abstracts and roentgenograms from the records of several patients whom I have observed, the trend of which supports the statement made by Ryerson that "encasement in plaster-of-paris actually produces bone atrophy and definitely delays healing."

In considering the end-results, I have used three terms: good, fair and bad, which should be defined. A good result is one in which a limp is not present or is only barely perceptible, hip movements are but slightly limited and there is no complaint of pain even after vigorous exercise. A fair result is one in which the limp is noticeable, there is considerable restriction of several of the hip movements and complaint of soreness and stiffness after exercise. With a bad result, all of the above signs are present to a more marked degree and, in addition, the patient is disabled by reason of pain and limited hip movements for heavier kinds of work or rougher sports.

I have selected fourteen cases which divide themselves into the following groups:

(1) True end-results, five cases: (a) without treatment, three; (b) with treatment, two.

(2) Patients in the late stage of the disease when the end-result may be predicted: four cases, all having had treatment.

(3) Patients still in the active stages and under treatment or observation: five cases.

Of the three patients in the first classification who had received no treatment, there was one with a good result and two with a bad result. The ages of these patients now are 26, 38, and 18 years. When these results are considered in relation to Flemming-Moller's rule for prognosis, we find an exception; for in the patient with a bad result the onset of the trouble occurred when he was only eight years old. According to Legg's theory, the prognosis depends upon the deformity of the head of the femur, but it is seen that one of these with a bad result had the mushroom type of epiphysis.

There were two patients with end-results who had received treatment. They are now 20 and 22 years of age and one has a fair and the other a bad result. In the case of the twenty-year-

old boy with a fair result, the onset of symptoms occurred in the eleventh year, and during his twelfth year he wore a caliper brace for six months with considerable relief of his symptoms. It is doubtful that this treatment materially influenced the course of his disease. His roentgenogram now shows the cap type of deformity of the femoral head. This result again refutes Legg's prediction because, with the cap type of deformity, he has a fair result; that is, the symptoms and restrictions are milder than Legg's article indicates. A good result after the onset of symptoms at the eleventh year forms an exception to the rule of Flemming-Moller. The other patient in this group at the age of 22 years showed a bad result, with a peculiar deformity of the femoral head which was difficult to classify. The neck of the femur was shortened, the head very broad with the outer portion projecting laterally some distance beyond the rim of the acetabulum, but flattened in an oblique plane. His trouble began when he was ten years old and, three years later, he was kept in a plaster spica for six months with great relief of the symptoms of stiffness and pain. According to Flemming-Moller's rule, his symptoms having appeared when he was only ten years old, we might have anticipated a fair result, whereas he is really incapacitated for heavier types of work.

In this small group of five cases with true end-results, we observe three with bad results, one with a good and one with a fair result. Two of the bad results were seen in the untreated group and one among those treated. It is admitted that in both patients who received treatment the measures were instituted so late in the disease that deformity of the head had doubtless already appeared.

The second group, with four cases, represents the late stage of the disease in which the character of the end-result may be reasonably anticipated.

In *Case 6*, the patient, now thirteen years old, has bilateral involvement, the trouble having started in the left hip when he was eight years old, and in the right a few months later. He has had no support or fixation and has taken vigorous exercise throughout the course of the disease. He now has no pain in the left hip even after strenuous exercise and very slight restriction of movements, but has moderate pain and limp associated with the right hip. The mushroom type of deformity appears in the late roentgenograms of both hips. The result will doubtless be fair. With bilateral involvement and without treatment, this patient's result conforms to the rules of both Flemming-Moller and Legg.

Case 7 of the second group is a boy now fourteen years old. The onset of the disease came in his fifth year, and twelve months later he put on a caliper brace and continued using it for a year, with some relief of his worst symptoms. He has a fair result and his roentgenogram shows the mushroom type of head deformity. His result should have been good instead of fair, according to Flemming-Moller. His treatment was instituted too late to expect it to change or protect the conformation of the epiphysis.

In *Case 8*, the third in group two, the beginning of the trouble occurred at eight years of age, and two months later the patient was fitted with a caliper brace, which he wore for three months. His acute symptoms then subsided, but he retained a moderate limp for about six months longer. Since then, although he has been riding, hunting, playing baseball and football, he has continued to improve, and now, at the age of thirteen, he has a good result and his roentgenogram reveals the mushroom type of epiphysis.

In *Case 9*, fourth of the second group, the onset of the hip trouble occurred when the patient was ten years of age; nine months later, her tonsils were removed and a plaster spica was applied for six weeks. When the plaster was removed a caliper brace was applied and worn for six months. She is now thirteen years old, has a noticeable limp, pain after moderate exercise, and is still disabled for heavier types of work. Her immediate result is bad and roentgenograms show the cap type of femoral head.

In this case, treatment served to relieve only the worst symptoms and she has progressed to a bad result. This would have been expected by the age rule of Flemming-Moller as well as by the epiphyseal deformity as indicated by Legg.

In this second group of four cases, we have seen one with a fair result, although the patient had bilateral involvement and no treatment, a second fair result in a young child with treatment instituted late, a good result with a short period of treatment with caliper brace instituted early in the course of the disease, and a bad result in a child of the same age with similar treatment begun nine months after onset of her symptoms.

The last group of five cases is still under observation. The immediate effects of the measures employed are reflected in the late clinical and roentgenologic examinations.

In *Case 10*, the earliest symptoms began when the patient was seven years old. Three months later, when his roentgenograms showed the initial stage of involvement, a tonsillectomy was done and plaster applied and worn for six weeks. His family was unable to purchase a brace and he began walking as soon as the plaster was removed. He is now eight years old, shows a marked limp, muscular spasm with adduction and flexion contracture; the roentgenogram shows the cap type of epiphyseal deformity with fragmentation still present, but calcification beginning to appear. He obviously needs more treatment and, although it is

Fig. 1

Case 12. Boy, aged ten years. Plate a (top left) made seven months after onset. Succeeding plates b (top right), c (bottom left) and d (bottom right) were made at the third, fifth and eighth months after application of a plaster spica and show rapid disintegration of the head.

now too late for such measures appreciably to affect the shape of the head, relief from full weight-bearing would doubtless overcome the muscular spasm and associated contractures.

In *Case 11* acute symptoms began at six years of age. When the patient was seen a year later his roentgenogram revealed fragmentation of the epiphysis. A caliper brace was fitted and worn for six months. Eighteen months later, the patient being nine years of age, he has a fair immediate result and a roentgenogram shows the mushroom type of epiphysis with calcification almost complete.

In *Case 12*, the first symptoms of hip disease were noted the tenth year. After one month, when his roentgenogram showed the initial stage of involvement of the epiphysis, the patient was kept in bed for short periods at irregular intervals and given heliotherapy. Seven months later he was put in a plaster spica and it was reapplied at intervals for eight months. Roentgenograms at different intervals during this period of fixation showed progressive disintegration and decalcification of the epiphysis and neck of the femur and acetabulum (*Fig. 1*). At the time when the process appeared at its worst, however, it was found that he had but slight tenderness and a fair range of free motion in the hip. The caliper brace was then applied and it was observed that the patient's symptoms rapidly improved and the range of his hip motions increased. Six months after beginning to use the brace he shows only moderate muscular atrophy and a very slight limp, and roentgenograms show almost complete recalcification of the epiphysis with but slight change in contour (*Fig. 2*). He is now twelve years old, the lesion is nearly healed and the immediate result is good. The epiphysis is of the mushroom type. The striking points in this case are the rapid appearance of bone atrophy while in plaster and the prompt recalcification with almost no distortion of the head when the patient was allowed up on a caliper brace.

In *Case 13*, symptoms referable to the hip were noted at the age of ten years. One month later, when the roentgenogram showed the initial stage of involvement, a caliper brace was applied and used for six months. At the end of this time, although the roentgenogram showed fragmentation of the epiphysis and no recalcification, all symptoms had disappeared and the patient was allowed to discard the brace. A month later he had a very slight limp, but

Fig. 2

Case 12. Rapid regeneration and recalcification were seen after the patient was permitted to be up on a caliper brace for six months.

Fig. 3

Case 14. Boy, aged three years. Plate a (top left), made ten months after onset when symptoms were active, shows the state of fragmentation. Plates b (top right) and c (bottom left), made three and four and one-half months later, respectively, illustrate progress while a caliper brace was being worn. Plate d (bottom right), made four and one-half months after leaving off the brace, indicates that calcification and regeneration continued after the brace was discarded.

no restricted movements and no pain, even after strenuous exercise.

Case 14 was a boy who began having hip trouble at the age of three. The condition persisted for ten months before a brace was applied and the roentgenogram then showed fragmentation of the epiphysis (*Fig. 3*). Within three months all symptoms disappeared and the range of motion was comp'ete; at the end of four and one-half months the roentgenogram showed slight recalcification. The brace was then removed and four and one-half months later (nine months after the brace was applied and nineteen months after onset of the disease) he has a barely perceptible limp, no restriction of hip movements and his roentgenogram reveals a head of almost normal contour which is rapidly recalcifying.

In the last group of five cases still in the active stage, we see one with marked symptoms and distortion of the epiphysis in which the treatment, consisting of fixation in plaster for six weeks, was obviously inadequate. There is another in which bone atrophy and decalcification appeared rapidly during nine months' fixation in a spica, but a good immediate result and prompt recalcification without deformity of the head followed within six months after the patient was permitted to move about on a caliper brace. Two other patients were permitted to discard their braces shortly after symptoms disappeared, even though recalcification was either not evident or had just begun, have good immediate results, and one (*Case 11*) shows a fair immediate result, although the brace was not applied until a year after the onset of symptoms when fragmentation was evident.

SUMMARY

(1) Abstracts of two articles on end-results of coxa plana are cited, together with some references to treatment from current text-books.

(2) A resumé of the records of fourteen cases of coxa plana is presented with special reference to the age of onset of the disease, the time and character of treatment rendered and results obtained.

(3) While the majority of the groups designated as true end-result and late stage cases followed closely the prognostic indications outlined by Flemming-Moller and by Legg, there were several definite exceptions to both rules.

(4) The group of patients still in the active stage of the disease permits of a better analysis of the immediate effects of treatment. While their number is too small to justify definite conclusions, the following thoughts are suggested:

(a) Fewer bad results are seen among treated cases.

(b) Absolute fixation in plaster is probably

unnecessary and, when employed, atrophy of the head and neck of the femur rapidly results.

(c) The use of a caliper brace, which serves to relieve the hip joint of full weight-bearing, and partially to restrict its movements, suffices to relieve the severer symptoms in all cases. When it is applied early in the disease, it appears to protect the softened femoral head from becoming flattened and distorted.

(d) The brace was discarded shortly after symptoms became quiescent without recurrence of acute symptoms. In certain cases, little or no calcification was apparent in the roentgenograms when the patient began full weight-bearing. Nevertheless, recalcification occurred promptly, with minimal distortion of the epiphysis.

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DISCUSSION (*Abstract*)

Dr. Robert W. Johnson, Jr., Baltimore, Md.—Dr. Caldwell's paper suggests, first of all, the wisdom of a general check-up of end-results of this condition in a large number of children's orthopedic clinics. A united effort might do much to enlighten us.

He has brought our roseate but hazy prognoses of this condition down to brass tacks. A psychological factor which has tended to make us unduly optimistic has been the relief we all feel when a child with an early hip limp turns out to have a coxa plana and not tuberculosis. We are so happy that we forget to take the lesser condition seriously, and yet we should, as Dr. Caldwell has shown us. There are several factors concerning prognosis on which Dr. Caldwell can shed some light. I wonder whether he feels that the age of onset is of primary prognostic importance? My own indefinite impression is that the younger the child the better the prognosis. Is duration of symptoms before treatment a great factor? I am inclined to attach considerable importance to this. How does the typically cap-type change compare with typical mushroom change in the head and affect treatment and prognosis? What role do the acetabular changes play in influencing ultimate function? What does the thickening of the femoral neck mean, except as a diagnostic sign? Is the slipping femoral epiphysis of the puberty period a variation of this condition? What is the ideal of treatment for a case in the active stage of the condition? My own practice has been to put early cases to bed, without traction or immobilization, and then graduate to a high-soled shoe on the good side and crutches, and then very gradual resumption of weight bearing with careful check-up of clinical signs of muscle spasm, increase of limp, and so on, as more important even than x-rays, since they seem to show up more rapidly than bony x-ray change. I scarcely ever use traction, and in recent years never immobilization in plaster, which I take to be in agreement with the speaker.

Dr. James Thomas Tucker, Richmond, Va.—It was my recent pleasure to visit Dr. Jacques Calvé's clinic at Berck Plage, France. The purpose of my visit was to get his opinion of the diagnosis and treatment of coxa plana. He showed me many x-rays of this condition and outlined his past experience in handling the cases. For a long time and with a large number of cases he treated the condition conservatively, that is, with fixation and all protective apparatus that would keep the patient from weight bearing and would secure immobilization. Another series of cases he treated symptomatically, and the end-results of these were as good as, if not better than, those treated conservatively. So his treatment is not to fix the hip except when spasm and pain are present, and to allow weight-bearing when the pain subsides and muscle spasm has disappeared.

He described and showed me x-rays of a similar condition in the bodies of the vertebrae where fragmentation occurred. This is known as Calvé's spine.

THE RELATION OF GALLBLADDER DISEASE WITHOUT JAUNDICE TO BRADYCARDIA AND HEART DISEASE: CASE REPORT*

By JOHN L. CARMICHAEL, M.D.,
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The following case is reported because of its possible bearing upon the problems of the nerve supply of the gallbladder and because of its possible bearing also upon the relationship between gallbladder disease and heart disease.

J. L. M., a white man, aged 28, was first seen on April 11, 1930, at about 8:00 a. m., in his home. The history was obtained that shortly after breakfast he had been seized with an acute abdominal pain. He had vomited a usual breakfast of eggs, toast, and so on. No history of previous indigestion or other attacks such as this could be elicited either from the patient or from his family.

He was found in intense pain, rolling and tossing in bed with his arms wrapped around his knees and his knees pulled up to his chest. The muscles of his abdomen were intensely rigid. His facial expression was frightened and agonized. There was no suggestion of shock. A hurried and rather unsatisfactory examination revealed a heart rate of about 40 per minute. The beats were regular. A blood count made at that time showed approximately 25,000 white cells.

He was sent immediately to the hospital, being admitted there at 10:00 a. m. with a pulse of 42 per minute and a temperature of 98°. Blood count at

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that time showed 26,300 white cells with a differential count of lymphocytes 5 per cent, large mononuclears 2 per cent, neutrophils 91 per cent, and myelocytes 2 per cent.

A catheterized specimen of urine at that time showed a few red blood cells. Otherwise it was negative.

Operation was decided upon. Although no definite diagnosis was made, it was believed likely that there had been a rupture of a viscus, probably the stomach or duodenum.

The operation was begun under local anesthesia and finished under nitrous-oxide-ether. The stomach, duodenum, and large and small intestine appeared normal. The gallbladder was small and of normal bluish color. The gallbladder was so firmly contracted upon its contents as to give the impression of being stony hard. It could not be emptied on pressure. There was a rather small band of adhesion between the duodenum and gallbladder near the cystic duct. There was little or no free fluid in the peritoneal cavity. Since it was believed best to subject the patient to the least possible trauma or anesthetic because of the heart condition, the gallbladder was drained and the abdomen closed after an unsuccessful search for gallbladder or cystic duct stones. There was thickening of the tissues of the cystic duct near its junction with the gallbladder. The gallbladder walls appeared normal in thickness and healthy. The operation was begun at 11:40 with pulse rate of 46 per minute and finished at 12:40 with pulse rate of 66 per minute. At no time during the operation was the pulse found to be above 74 per minute.

The pulse gradually increased in rate after the operation until on the fifth postoperative day it reached 72 per minute. It continued regular.

On the second postoperative day the first drainage of bile through the drain tube occurred. It was bright green in color and clear.

The remainder of the convalescence was uneventful.

The patient remained apparently well until July 20, 1930. On this date he was seen again in an attack of acute abdominal pain. It had begun in the epigastrium, localized definitely at the lower end of the sternum, and had spread to all the abdomen and chest. He vomited profusely at first. The vomitus contained a little bile. His temperature was 98° and his pulse rate was again 42 per minute. He was given 1/100 grain of atropine and 3/8 grain of morphine to be followed later by a laxative. On July 21, the next day, the patient came to my office. His pulse then was 78 per minute and regular. The apex beat was in normal position, his heart sounds were normal and he appeared well.

The patient reported on October 15, 1930, over the 'phone that he was doing well and had had no more attacks. On October 23, however, he was seen in an attack similar to the others, but not so severe. He vomited considerable bile. His pulse rate was 60 per minute. A hypodermic of morphine and atropine again relieved him.

In the interval between these attacks and in the period of time since the last attack the patient's condition has been followed closely, he has at all times

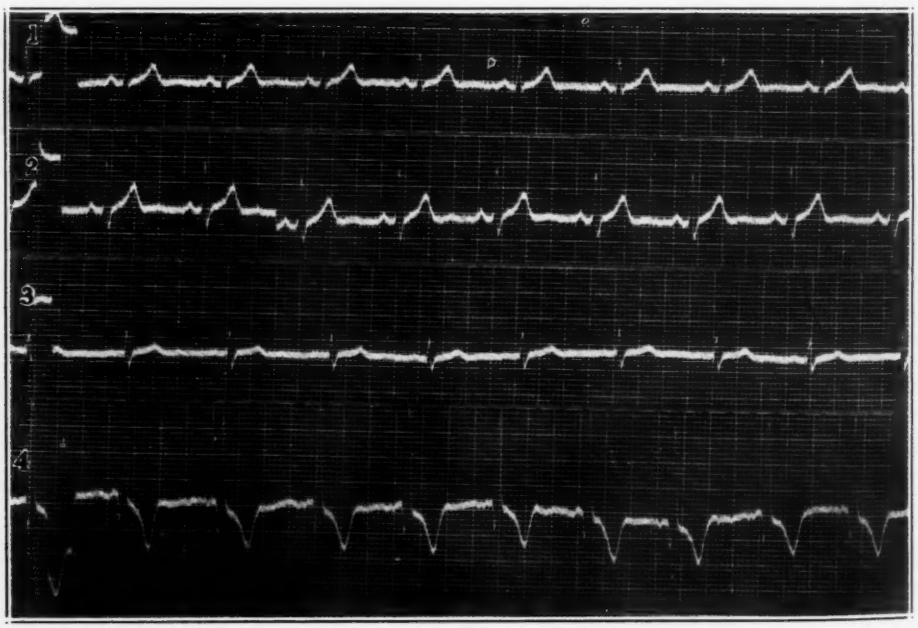

Fig. 1
Electrocardiographic tracings in the four leads, (1), (2), (3) and (4).

seemed a well man, and has carried on his work as inspector in the steel mills without inconvenience. Diet designed to reduce the chance of gallbladder colic and saline laxatives, especially sodium phosphate, have been the treatment during this period.

The interpretation of the pathologic condition involving the gallbladder is that the wall of the cystic duct had become inflamed and swelled, thus closing the lumen of the duct. Food taken into the stomach caused contraction of a healthy gallbladder muscle which developed great pressure and thus produced violent pain, which only the hypodermic of morphine and atropine relieved. At no time was there noticeable jaundice or other evidence of common duct obstruction. No estimation of serum bilirubin, however, was made.

An explanation of the bradycardia which suggests itself is as follows: there are some afferent vagus fibers to the gallbladder, or at least to the cystic duct, whose irritation by violent pressure caused overflow impulses to neurones of the vagus which supply the inhibitory impulses to the heart, causing thus a reflex slowing of the heart by partial heart block. This explanation, it will be noted, presupposes the presence of afferent vagus fibers to the gallbladder or cystic duct which is denied by Kuntz.[1] It is believed, however, that the latter author would admit that it has not been fully proved that afferent vagus fibers are absent from the gallbladder.

To those who would oppose to this explanation the occurrence of many cases of gallbladder colic without bradycardia, the suggestion is offered that this is an unusual case in that there exists a normal gallbladder wall and muscle and indeed probably a normal biliary system except for the disturbance in the cystic duct. This would explain why a small stony hard gallbladder was found. It probably takes that unusually intense pressure induced by a healthy gallbladder muscle to produce an overflow of nerve impulses to the neurones supplying the heart, at least an overflow sufficient actually to slow the heart. There might be subminimal reflex overflow impulses which would reach the heart by way of the vagus without slowing the heart.

A further suggestion is that perhaps those cases in which bradycardia occurs with jaundice are cases in which the reflex action on the heart occurred through the vagus nerve fibers and that jaundice had nothing to do with the bradycardia, as some reports in the recent literature indicate.

There seems to be a fairly general impression among internists that gallbladder disease has a deleterious effect on the heart out of proportion to the effect that an ordinary focal infection would produce. This impression implies that the removal of the diseased gallbladder in cases showing heart disease and gallbladder disease may give a decided improvement in the heart condition, and this, it is believed, is a result confirmed by the internists. If that is true, may not that circumstance be due to certain deleterious reflex stimuli from the gallbladder to the heart.

This does not seem to be a novel theory when it is recalled that disease of the gallbladder is considered to be a common cause of reflex disturbance of gastric function.

Since it was felt that if this case report were to have the greatest value, intrinsic cardiac disease would have to be ruled out, Dr. H. R. Carter was asked to study the heart condition and make electrocardiographic tracings. Parts of the graphs are given here, as taken on July 19, 1933, together with Dr. Carter's report, which reads as follows:

"*Electrocardiogram.*—Rate 80. Rhythm-sinus arrhythmia, slight. Axial direction, normal. P 1, 2, 3, positive. Height 1.5 mm. Duration, 0.08 second. P-R interval, 0.16 second. QRS complexes, duration 0.08 second. R1, 5.5 mm. R2, 8.5 mm. R3, 5.5 mm. S3, 2.5 mm. T 1, 2, 3, positive. Height, 4 mm. QRS-T, duration 0.34 second. S-T plateau, slightly above iso-electric level in Leads I and II; definitely above in Lead III, and below in Lead IV.

"*Interpretation.*—The departure from the normal of the S-T plateau in Lead IV is suggestive of impaired coronary circulation.

"The changes in Lead III suggest myocardial disease, not definitely coronary in origin."

SUMMARY

(1) A case of cystic duct obstruction with strong contraction of gallbladder muscle associated with a marked bradycardia is herein reported. The attack with the associated bradycardia, judging from clinical signs and symptoms, has recurred twice since the original attack.

(2) Electrocardiographic study approximately three years after the attacks suggests possible intrinsic cardiac abnormality, although the pa-

1. Kuntz: The Autonomic Nervous System. Lea & Febiger, 1929.

tient's subsequent history would not suggest cardiac disease.

(3) This case report is offered with the hope that there may develop some interest in the study of the relationship of cardiac disease to gallbladder disease. The writer realizes, however, that the possibility of intrinsic cardiac disease weakens the hypothesis offered here in regard to this relationship.

ANGINOID SYMPTOMS OF GALLBLADDER DISEASE*

A SURVEY OF THE LITERATURE

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Since early in this century it has been recognized that at times there is a relationship between cardiac manifestations and gallbladder disease. Kerr, in 1904, reported a case of an acute anginoid attack in a patient presenting the symptoms of gallbladder disease, but he believed both syndromes due to a disturbance of protein metabolism. In 1907, Riesman discused the subject with special reference to the appearance of murmurs during jaundice and thought there were two responsible factors: (1) myocarditis due to the accumulation of biliary salts and acids in the blood, and (2) the actual pain of the gallbladder colic. In 1909, Babcock reported three cases of cholecystitis with anginoid pain, in two of which an erroneous diagnosis of angina had actually been made, and a decade later he discussed at length the relationship between gallbladder disease and cardiac dyscrasia. Lichty, Willius, Leech, Roberts, Yoell, Brown, Miller and others have studied the subject and reported cases and conclusions. Despite this growing literature, apparently the rank and file of the profession have realized neither the significance of the relationship nor the necessity for careful examination and evaluation of both organs when either presents symptoms.

Just the method of action by which a diseased gallbladder produces heart derangement has been the subject of considerable speculation. One very reasonable theory is that the presence in the circulating blood of an increase of bile salts, bile pigment, or other substances incident to repeated acute attacks is toxic to the heart muscle. Another is that the infected gallbladder acts as a focus for the elaboration of bacterial toxins. Probably both theories are in a measure correct. Practically all observers agree that the actual pain in anginoid gallbladder cases is due rarely to actual inflammation, or much more often to the expulsive efforts made to rid the gallbladder of a stone or a mucoid plug, and that it actually represents a biliary colic with the pain radiated precordially along the usual anginal paths.

The mechanism of the radiation is explained by the innervation of the heart and the gallbladder. While observers are not in entire accord, it is probable that the nerve supply of the latter viscus is derived from the right phrenic, the left vagus, and from the splanchnics through the celiac plexus, the fibers of which arise from the cord from the sixth thoracic to the first lumbar nerves. Both motor and inhibitory fibers enter by way of the splanchnics and sensory fibers by both the vagus and splanchnics. The phrenic distribution to the gallbladder is questioned by many, but certainly the pain radiation would indicate the correctness of Mayo's suggestion that this nerve must at least in some way be associated with the cholecystic nervous mechanism, and further evidence is furnished by the radiation of pain to the shoulder by actual irritation of the nerve during a phrenicectomy operation. The heart receives fibers, essentially or probably entirely inhibitory, from the vagus; others, largely accelerator, from the sympathetic, the filaments of which pass through the inferior cervical ganglion. The posterior cardiac plexus receives from the superior cervical ganglion the superior cardiac nerve which is believed to be entirely motor; the middle and inferior cardiac nerves from the corresponding cervical ganglia, both of which are known to conduct both afferent and efferent stimuli; and fibers from the first to the fifth dorsal thoracic nerves through the white rami communicantes and the corresponding dorsal sympathetic ganglia. Pain from the heart is conveyed to the sympathetic system along fibers in the middle and inferior cardiac nerves to the corresponding cervical ganglia and over fibers from the posterior cardiac plexus to the upper five thoracic ganglia. The painful stimuli are transmitted to the posterior roots of the cord over the white rami, and as there are none of these in the cervical area, such pain sensations as go over the middle and inferior cardiac nerves must be relayed through the up-

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per five dorsal nerves which have the white communicant rami. With this innervation in mind, it can be seen that the viscerosensory reflex phenomenon accounts for the distribution of the pain.

The cause of anginal pain has been a moot question since the days of Heberden. The term "angina," as usually understood, includes a triad of major symptoms: (1) the pain, usually severe and lancinating with radiation to the left shoulder and down the left arm; (2) the sensation of constriction or stifling, and (3) the fear of impending death, some or all of which may be wanting in any individual case. As Roberts points out, instead of being considered a separate disease entity, angina should be looked upon as a painful symptom complex which may be produced by several different pathological states. With this enlarged concept of angina, we can understand how extracardiac causes can give rise to a presenting anginoid picture and, when the gallbladder nerve supply is considered, how disease of that viscus especially may initiate it.

The frequency of association of gallbladder and heart disease is not agreed upon by all observers. Brown says that gallstones are about twice as frequent in cardiopaths as in normal individuals in the same age group. Tennant and Zimmerman, in a series of 1,600 consecutive autopsies, 694 of which were on subjects over forty years of age, found 126 cases of arteriosclerotic heart disease, of which 33 showed coexisting gallbladder disease. They believe that there is a significant association between heart disease in general and gallbladder disease. Rehfuss studied 200 consecutive cases of gallbladder disease and found that in 71 cases, or 35.5 per cent, there was evidence of cardiac consciousness in some form. In 6, or 3 per cent, the pain seriously suggested angina. Willius and Fitzpatrick reported 596 cases of chronic disease of the gallbladder, of which 229, or 39 per cent, had organic disease of the cardiovascular system. Five cases of angina were noted, 0.84 per cent of the entire series. Schwartz and Herman reported 109 cases of gallbladder disease with 63 per cent showing some form of heart manifestations, while in a control group of an equal number of non-cholecystic patients within the same age limit, only 41 per cent showed abnormal heart findings. They do not record the number of anginas in the series. Leech studied 116 consecutive cases of proven non-malignant gallbladder disease, and concluded that whatever the apparent relationship between the two conditions, the actual heart disease is due to obesity, aging processes, or other causes rather than to cholecystitis. We quote: "The general conclusion is that there is no definite relationship between gallbladder disease and disease of the cardiovascular system." Lichty reports a series of 18 cases with heart manifestations which he believed to be sequelae of coexisting gallbladder disease. Of these five were anginoid cases, three of which were operated upon and cured. Without giving exact figures he says these cases were culled from approximately 1,500 gastro-intestinal syndromes, about 600 of whom had either appendectomies or cholecystic operations. Akana, Greely and Farr report 424 cases of gallbladder disease with special reference to the distribution of the pain. In only three instances was the radiation to the chest and in only one of these to the actual cardiac area. Kahn and Barsky reviewed 200 cases of angina and found 10 of them with associated gallbladder disease. Yoell reports 63 cases of cholecystitis, three of which presented a definite anginoid picture, 4.76 per cent. Many cases have been reported of pain with typical radiation down the left arm, and Brown, Miller and Roberts report instances with angor animi relieved entirely by operation. A sense of constriction accompanying the pain is very rarely present, however. In fact, in the literature we were able to locate only one instance so described and in two others the pain is characterized as "gripping." This feature of the syndrome, which must have influenced Heberden strongly when he chose the name "angina," may not be frequent in the gallbladder type because it really is a visceromotor phenomenon representing a spasm of the intercostal muscles, whereas the pain is a viscerosensory reflex, and the angor animi a fear reaction. At any rate we may conclude that there are cases of the anginoid syndrome dependent upon gallbladder disease and relieved by surgical removal of that viscus. The actual proportion of such cases is small, varying in different series reported, but probably less than 1 per cent of all gallbladder cases. In studying any individual patient, the fact must not be overlooked that both biliary colic and an anginoid syndrome, cardiac in origin, may coexist in the same patient. In fact, MacKenzie points out that this was true with John Hunter and suggests that from Hunter's own description only in the last years of his life were his painful attacks really anginal, the earlier painful episodes showing the character-

istics of gallstone attacks. At autopsy coronary sclerosis was found, as Jenner had diagnosed, and gallstones as well.

Given, then, a patient with the anginoid syndrome, how are we to determine the causative pathology? First a careful history is essential. The patient should be allowed to tell his story in his own way and then obscure details may be clarified by careful questioning. Proper evaluation of the symptoms will be of great aid. Pain initiated or accentuated by effort, emotion, or exposure to cold suggests heart disease, and its radiation down the left arm less forcibly so. Angor animi strongly suggests a cardiac origin and a sensation of constriction almost always points to the same conclusion. A precordial sensation of tenderness or soreness and a feeling of heaviness or soreness in the left arm following an attack indicate rather a cardiac basis. The pain of cardiac origin is usually more lancinating, of shorter duration, and more readily amenable to rest than that due to the gallbladder, but both types are subject to wide variations. In general terms, women are more prone to the gallbladder type and men to the cardiac. A history of preceding intolerance for certain articles of food points more to the gallbladder, as does vomiting with the attack, but the digestive symptoms are manifold and overlap most confusingly. The upper social strata tend more to the cardiac and the lower to the cholecystic. Age above fifty suggests cardiac disease; below, cholecystic. Jaundice is practically diagnostic of biliary disease, and in general the longer the duration of premonitory symptoms, the less the probability of cardiopathic origin of the pain. Relief of pain by nitroglycerin usually means that the origin is cardiac, but this is not always true, for this remedy has been known to relieve the biliary type.

Careful physical and laboratory examinations usually aid in arriving at a correct diagnosis. Tenderness on pressure over the left eyeball is said to be present only in cardiac cases. Tender points over the left shoulder and precordium incline us toward the heart; over the right shoulder, over the gallbladder area either front or back, and in the right midaxillary line between the ninth and tenth ribs, toward the gallbladder. At times tenderness can be brought out better by percussing the flexed finger, placing the tip directly over the gallbladder. A hypersensitivity over the right lower back in the costovertebral angle was found by Levene in 92.3 per cent of cases showing x-ray evidence of gallbladder disease. This is found over the course of the tenth and eleventh intercostal nerves and is elicited by placing the patient in the ventral prone position and stroking simultaneously the two sides of the back from above downward just internal to the vertebral border of the scapula. Increased tensity of the right rectus muscle is not found in the cardiac cases. A patient with biliary colic, when fluoroscoped during the attack, shows a definite restriction of the excursions of the right diaphragm. The icteric index may aid. The electrocardiogram may be within normal limits and the angina still be cardiac. Leech studied 53 of his cases carefully from an electrocardiographic standpoint and found that 70 per cent had a definite left axis deviation. Levogram, however, is to be expected in cholecystopaths because of (1) the age group in which they fall; (2) the physical type of most of them; and (3) their tendency to obesity. Cholecystography is a *sine qua non*.

Nowhere in the whole realm of medicine is there greater necessity for close cooperation between internist and surgeon than in the handling of these patients. Ordinarily we believe that all gallbladder cases should be studied medically before operation, and especially is the heart to be evaluated. Likewise if there is any question of a biliary basis for abnormal heart findings, surgical consultation is to be had because not only do these patients stand surgery well, but usually the results of operation are brilliant indeed.

SUMMARY

An anginoid syndrome may be dependent upon cholecystic disease, but occurs in probably less than one per cent of all pathological gallbladders. Pain with fairly typical radiation is the commonest of the major anginoid symptoms; angor animi is fairly often present; but sensation of constriction is exceedingly rare. The minor anginoid symptoms are so variable as to be of little value in diagnosis. Tender left eyeball, tender spots on the shoulder, chest wall, or over the gallbladder, are of definite diagnostic value. Jaundice and increased right rectus tensity are practically pathognomic. The electrocardiogram may or may not aid, but cholecystographs will usually clinch the diagnosis. In any given case a complete diagnostic study

should be done and a surgeon called in consultation. Unless strong contraindication exists, prompt surgery should be resorted to.

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THE CLINICAL VALUE OF THE STUDY OF THE PERIPHERAL CIRCULATION*

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The purpose of this paper is to discuss the methods that are available to the practicing physician for the study of diseases of the peripheral circulation. The comparatively recent introduction and extended use of accurate procedures of examination have led to a much more complete understanding of the pathology of this group of diseases than previously existed. Studies in this field of medicine received their modern impetus from Buerger's now classic work on thrombo-angiitis obliterans. The fundamental studies of Buerger[1] and of such men as Lewis,[2] Starr,[3] Morton and Scott[4] and Samuels[5] have led to the development of clinical methods that have not only diagnostic but also definite prognostic value.

Before describing and discussing the more technical aspects of this subject, it should be emphasized that here as elsewhere in medicine the actual history and physical examination of the patient are of the utmost importance. There is as yet no diagnostic test which makes accurate history taking and careful examination unnecessary.

There are certain symptoms and physical findings which should immediately call attention to the possibility that peripheral vascular dysfunction is present. Pain is the most prominent of the symptoms. The type of pain which is almost pathognomonic of arterial disease of the extremities is that known as intermittent claudication, a sharp, cramp-like pain in the calf muscle or, more rarely, on the plantar surface of the foot, which is always induced by exercise and relieved by rest. This pain is probably the result of muscle anoxemia and is caused by excessive use of the muscle in the presence of a deficient blood supply resulting from arterial spasm or organic occlusion. The

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pain of intermittent claudication is always in a muscle, most frequently the calf, and although it may be close to, it is never in a joint. The pain due to varicose veins, on the other hand, is usually superficial and frequently follows the course of either saphenous vein.

Although pain is usually the symptom which causes the patient to seek medical advice, occasionally his anxiety is first aroused by coldness of the extremities or a change in color of the part, and frequently these symptoms are present in association with pain. A sense of coldness may be present long before pain is felt and its onset during adult life should always arouse the suspicion of arterial disease. Ordinarily, however, it is the examining physician who first notices the change in the temperature of the affected part. It is probably not possible to detect differences of less than two degrees with the back of the hand. For usual clinical purposes, however, greater refinement is hardly necessary, although with special instruments such as a thermocouple accurate determinations can be made. It must be borne in mind that disturbances other than obstruction of the arterial supply may be responsible for a lowering of the skin temperature. There may be a dysfunction of the vasomotor nerves such as is occasionally seen in various neuropathies, although it must be acknowledged that the pathogenesis of this symptom in these conditions is still obscure.

The color changes in the skin of an involved limb may be striking. The most significant of these is marked pallor when the limb is in the horizontal position, mottling, or such abnormal colors as undue redness and cyanosis. Pallor is the result of constriction of the superficial vessels. It is seen in association with arterial occlusion only when this occurs suddenly and it then lasts until there has been some dilatation of the superficial vessels in an attempt to establish collateral circulation. Pallor is ordinarily not seen in slowly developing arterial occlusion for this reason.

Much may be learned about the state of the circulatory efficiency of an extremity by the application of two simple tests based upon changes in color.

The patient is placed in the recumbent position and the limb elevated to the vertical. Marked blanching does not occur normally. If it does occur it is indicative of excessive compensatory constriction of the superficial capillaries and the degree and speed of appearance of the blanching are in proportion to the involvement of the cutaneous circulation. After blanching has occurred the limb should be lowered quickly to the dependent position. If rubor or rubor and cyanosis appear the deep vessels are probably diseased. What Buerger has called "the angle of circulatory sufficiency" is determined in a somewhat similar way. This test is based on the fact that the normal limb will retain most of its color when raised from the dependent to the upright position. With vascular dysfunction, as just noted, blanching occurs as the limb is moved upward. Once blanching has taken place, the limb is slowly lowered until the normal color or, as is generally the case in the presence of circulatory disease, rubor and cyanosis just begin to reappear. The angle from the vertical at which this is seen is known as the angle of circulatory sufficiency and the greater this is, or, in other words, the lower the limb has to be placed before the color change occurs, the greater is the degree of circulatory involvement.

This test is not only helpful in diagnosis, but is also of assistance in following the progress of the disease. Occasionally, however, because of vasomotor irritability, blanching persists for some time and therefore interferes with the accuracy of the examination.

Especially with slowly developing arterial occlusion definite trophic changes may be noted in the skin of the affected extremity. These are particularly a thinning of the skin, a shriveled appearance due to the loss of subcutaneous fat and a loss of the normal softness and oiliness of the skin. In addition there may be thickening and brittleness of the nails, especially that of the great toe. These changes have often seemed to be the premonitory signs of the dry gangrene and mummification which occur in long-standing and advanced arterial occlusion. They are seen only when the vascular disease develops quite slowly. When the process is more acute they rarely have time to occur before the onset of gangrene.

The appearance of gangrene in an extremity is always of serious import, as it is an indication that circulatory disease is so marked that the blood supply has fallen below even the minimal amount necessary to keep the tissues alive.

When gangrene is due to occlusion of the vessels the condition is frequently unilateral or if bilateral is not symmetrical; while in disturbances of vasomotor origin the gangrene tends to be both bilateral and symmetrical. Gangrene due to vasomotor disorders is also more likely to involve the more superficial tissues, while occlusion of the nutrient vessels causes damage to all the tissues of the affected part and if allowed to progress will finally end in spontaneous amputation. Gangrene is not necessarily a widespread destruction. In fact, it is extremely important to recognize the tiny gangrenous spots which occur around the bases of the nails in relatively early vascular disease. If the serious

import of this comparatively slight tissue death is grasped many patients can by proper treatment be spared tragic progress of the damage, especially since it is now generally admitted that the majority of early cases can be satisfactorily managed by conservative medical measures without resort to radical surgery.

A physical finding of great significance associated with that important vascular disease, thrombo-angiitis obliterans, is migrating thrombophlebitis. Indeed, it may be said that the occurrence of this type of phlebitis with evidence of arterial occlusion is a combination practically pathognomonic of Buerger's disease. In a typical case there occurs along the course of one or more of the superficial veins, most frequently the internal saphenous, small indurated patches which vary in diameter from 1 to 2 cms. They are usually quite tender and may be adherent to the skin. Such attacks of phlebitis may last a week or more and then clear up, to be followed shortly by a recurrence higher in the same vein or possibly in an entirely different one. As the lesions grow older the veins may feel like dense cords and the adhesions that form around the inflammatory masses may involve the accompanying arteries and nerves. The occurrence of migrating phlebitis should always arouse a suspicion of obliterating arteritis, although the arterial disease may not be present when the phlebitis is first seen.

In studying the peripheral circulation it is extremely important to palpate the accessible arteries. Absence of pulsation is fairly reliable evidence of occlusive disease. In the lower extremity of a normal individual the pulsations of the femoral, popliteal, posterior tibial and dorsalis pedis arteries are usually easily felt. If the popliteal is not easily palpated because of obesity or failure to relax the hamstring muscles the technic suggested by Buerger may be of great assistance. The patient is placed on the abdomen and the knee flexed. If the examiner raises the leg from the table by lifting from the ankle and the patient relaxes the artery can usually be felt.

The use of the x-ray in the study of the peripheral vessels is of an importance that is distinctly secondary to that of other methods of examination. Generally speaking, this procedure is of value for two reasons. Through it some idea of the extent and degree of calcium deposition can be obtained and the occurrence of bone rarefaction can be determined. The degree of vascular calcification is not of first significance, since calcium deposit is a late phenomenon in the arteriosclerotic process and in some arterial diseases such as thrombo-angiitis obliterans does not occur at all.

Of the more exact technical methods used in the study of the peripheral circulation the two most generally employed are the determination of oscillometric readings and the histamine test. The oscillometer was first used by Pachon[6] in 1909. An inflated cuff is applied over the arteries at various levels. The oscillations of a connected needle register the pulsations in the vessels. The degree of oscillation at any given level of an extremity is known as the oscillometric index at that level. In Samuel's experience the average normal readings are:

Upper arm	4.0 to 20.0	Thigh	4. 0 to 16.0
Elbow	2.0 to 12.0	Below knee	3.0 to 12.0
Wrist	1.0 to 10.0	Ankle	1.0 to 10.0
Palm	Faint to 2.0	Foot	0.5 to 2.0

In Samuel's series few normal individuals gave less than a reading of 1.0 at the foot. We have found, however, that many normal adults give readings as low as 0.5. The wide range of the normal figures might appear as an objection to oscillometry and it is true that incipient arterial disease may escape notice because of this fact. It must be realized that in addition to the factor of arterial distensibility, knowledge of which is being sought by the oscillometer, such items as the stroke volume of the heart and the amount of muscle and fat overlying the vessel influence the reading. The influence of these disturbing factors can be avoided to a certain extent by comparing the readings at the same level in both legs or both arms. If the reading is definitely lower in one extremity than the other, arterial disease may be considered to be present even though the lower reading is above the normal minimum. Our observations lead us to feel that the consideration of the symmetry of the readings is as important as the readings themselves.

Oscillometry is intended primarily for the study of the deep arterial circulation. It gives little or no information about the collateral or superficial vessels. It indicates how much the artery or arteries at the level at which the cuff is applied are able to distend with each systole of the heart. By means of the oscillometer it is possible to determine whether a given artery is pulsating at all and, in cases in which embolism or thrombosis has occurred, to locate the level of the occlusion. Its practical value is best illustrated by citing a few cases in which it was used.

E. G. was a diabetic, aged 67 years. Six years be-

fore admission she had had an ulcer on one of the toes of the right foot which had healed slowly. Nine months before admission she began to have severe pains in the left foot, particularly in cold weather. Exertion caused shooting pains in the left leg and she was greatly troubled by a feeling of coldness in the feet. On admission she had beginning gangrene of the right big toe and rather advanced gangrene of all of the toes and the distal part of the left foot. The oscillometric readings were:

	Right	Left
Ankle	zero	0.1
Below knee	zero	0.75
Above knee	0.5	1.5

These readings indicated marked impairment of the arterial supply to both legs, particularly the right, with practically complete obstruction at about the popliteal level on the right side.

Another diabetic, M. K., aged 52 years, was admitted with advanced trophic changes of the left great toe, gangrene of the upper surface of the right great toe and dependent rubor up to the middle of both feet. The oscillometric readings were:

	Right	Left
Foot	zero	zero
Ankle	faint	faint
Below knee	0.75	1.5

That arterial occlusion was quite extensive was evident from these readings and amputation of both legs above the knees finally became necessary.

The oscillometer is of value in localizing the site of sudden arterial occlusion such as occurs in embolism.

A white man, aged 65 years, was admitted with hypertensive heart disease and moderate decompensation. About 10 days after admission he developed a right-sided pleural effusion and the following day was found to have partial heart block which persisted for three weeks. During this time the nurse noted that she was unable to obtain a pulse reading at the right wrist. The oscillometric readings then taken were:

	Right	Left
Above elbow	12.0	12.0
Below elbow	zero	6.0
Wrist	zero	4.0
Palm	zero	2.0

The extremities were of practically the same temperature to touch and there were no color changes or symptoms of any kind referable to the arm. In four days the oscillometer showed faint pulsation in the vessels of the lower right arm and the radial pulse became questionably palpable. The patient died one month later of heart failure and without developing any gangrene.

Oscillometric investigation may also be of assistance in ruling out the presence of circulatory disease when this is considered among the diagnostic possibilities. Thus in a patient with pains in the legs the readings were:

	Right	Left
Foot	0.5	0.5
Ankle	1.5	1.5
Below knee	4.5	4.0

Although these readings indicated that a moderate degree of arteriosclerosis might be present they were not strikingly abnormal and a further study of the case revealed that the pains were due to tabes.

Histamine was first used by Lewis in his studies of the capillaries, but was originally applied clinically by Starr. Its use is of value chiefly in determining the state of efficiency of the superficial circulation. The substance used is histamine acid phosphate in a solution of 1 to 1,000 in distilled water. The procedure is that suggested by Starr.

The patient is placed in the horizontal position with the toes pointed inward to prevent the histamine solution from running over the dorsum of the foot. The upper surfaces of the feet and the anterior surfaces of the legs are cleansed gently with alcohol, which is allowed to dry. About 0.5 c. c. of the histamine solution is drawn up into a hypodermic syringe and one drop is placed on the dorsum of one foot. About ten intracutaneous punctures are made in rapid succession through the drop of histamine. The process is repeated on the dorsum of the other foot and in rapid succession in the mid-line at both ankles, external to the crests of the tibiae below the knees and in the mid-line just above both knees. With a little experience the eight tests can be performed in one minute. Readings of the reactions are taken at two and one-half, five, ten and fifteen minutes after the first intradermal injection and, if possible, all eight of the tests should be read within the length of time which was necessary for the injections. The reaction normally consists of two parts. There is first a zone of erythema due to arteriolar dilatation. This is called the "flare." In addition there is located in the center of this a wheal which is due to exudation of plasma. The diameter of both the "flare" and the wheal are noted at each injection site at each reading. Normally both the wheal and the "flare" begin to appear within five minutes and increase in intensity up to the fifteen minute reading and then begin to fade. The "flare" is usually about the size of a five-cent piece, but variations in the size of the reaction are not of diagnostic importance unless the reaction is so small as to be negligible. It also occasionally happens that only the "flare" or only the wheal appears. Along with most workers in the field we believe that this appearance of an incomplete reaction is not necessarily abnormal. From a diagnostic standpoint significance must be attached to failure of the reaction to appear at all or to appear only after more than five minutes have elapsed. The reaction is ordinarily more intense as one approaches the knee. This is not necessarily so, however, and not infrequently the reaction is less marked at the ankle than it is over the dorsum of the foot. This is probably indicative of the less well developed superficial circulation in the former region.

The histamine reaction is particularly useful in determining the imminence of gangrene in cases in which it is known that the deep vessels are occluded. Failure of the reaction to appear or its appearance only after an abnormally long waiting period indicates that the superficial

as well as the deep vessels are involved and gangrene is likely to occur under these circumstances. In advanced vascular disease, amputation should always be performed above the level of a normal response to histamine in order to be sure that there will be healthy tissue at the site of operation.

As an example of the typical histamine reaction in cases with impaired circulation the following case is briefly described.

A diabetic woman, aged 68 years, on admission showed beginning gangrene of one toe of the right foot and advanced gangrene of all of the distal portion of the left foot. Oscillometric examination showed much more occlusion of the deep vessels of the right foot than of the left. The histamine test showed, however, that although there was deficiency of the superficial circulation of the right foot the deficiency in this respect was much more marked on the left. Such findings explained the more extensive gangrene of the left foot.

In addition to oscillometry and the histamine reaction, methods for determining the temperature of the extremities are also of value in circulatory studies. The most important of these methods is the use of the thermocouple and galvanometer for the measurement of skin temperature. The thermocouple is an instrument designed to utilize the electrical current developed by the difference between the temperature of the skin and that of a standard. Unfortunately this method is time-consuming and therefore does not have the clinical applicability of the other technical procedure already mentioned.

The value of these methods of studying the peripheral circulation can best be summarized by presenting briefly two fairly typical cases in which they were used to advantage.

The first patient was a woman, aged 50 years, who had suffered right hemiplegia and aphasia one year before her admission, but had fully recovered from these. She was admitted complaining of severe pain and numbness in the calf of the left leg which had developed suddenly while she was walking up a flight of stairs. She had also lost the sense of pain perception in the left leg. On admission the left leg was discolored and was cold to touch. The thermocouple showed that the left foot was three degrees colder than the right foot at the toes, one degree colder at the arch and more than one degree colder over the dorsum. Histamine caused no reaction of any kind in fifteen minutes at the dorsum of the left foot or at the left ankle and below and above the left knee the reactions were feeble. The reactions occurred on the right foot and leg in slightly more than the normal time. Oscillometry gave these readings:

	Right	Left
Ankle	0.25	zero
Below knee	0.4	zero
Above knee	2.0	zero

The routine physical examination was essentially negative and the laboratory studies were normal. A diagnosis of complete arterial occlusion at the left groin and partial occlusion below the right knee was made. Two weeks after admission gangrene began in the left foot and later developed in the right foot. This process rapidly extended up both legs and at the same time hematuria was noted. At the time of death, four weeks after admission, the gangrene extended from the toes to the hip on the left and to the mid-thigh on the right. A partial autopsy showed that two inches above the bifurcation of the aorta there was an atheromatous plaque. At this point a rather dense thrombotic mass was attached to the left wall of the aorta. It was so large as almost to reach the opposite side so that the vessel was almost completely occluded, although there was still a channel between the thrombus and the right aortic wall. The upper border of the thrombus had extended above the opening of the left renal artery. That vessel was completely occluded and the entire left kidney was acutely infarcted. The common iliacs were completely occluded and the femorals were also completely obstructed by thrombi. It seemed probable that the process had had its inception at the site of the atheroma of the aorta.

The second patient is a Russian Jewish man, aged 36 years, recently admitted to the hospital complaining of pains in the feet and legs of three weeks' duration. These had developed while he was walking and had been so severe as to compel him to walk very slowly with frequent rests. The day after the pains began he awakened to discover that along the inner surface of the lower right leg there were numerous red and tender spots. The veins were prominent and quite red. The pains increased in the interval prior to admission so that even standing was apt to induce it. Rest, however, always brought relief. Shortly before he came into the hospital he noticed coldness of both feet. Examination of the extremities on admission revealed many interesting findings. Both feet showed marked dependent rubor, which was slightly more prominent on the right. When the feet were elevated there was excessive and rapid b'anching and the feet had to be lowered below the horizontal before color returned. Both feet were cold to the touch and there was a small spot of beginning gangrene at the base of the right second toe. Along both internal saphenous veins there was definite though subsiding thrombophlebitis which was irregularly distributed through the veins. The right dorsalis pedis was not palpab'e. Pulsation was felt in the left dorsalis pedis and in both posterior tibials and popliteals. The oscillometer readings were:

	Right	Left
Feet	zero	faint
Ankles	1.25	1.25
Below knees	3.0	4.0
Above knees	3.0	6.0

The histamine test was normal everywhere except over the dorsum of the right foot, where the reaction was markedly delayed.

The picture in this case from the standpoint of both the clinical evidence and the examination findings are those of arterial occlusive disease and there seems little doubt that the process is thrombo-angiitis obliterans. The positive histamine test in the right foot

is in keeping with the occurrence at that site of very early gangrene.

At the present time he is being treated for Buerger's disease and receiving intravenous injections of saline and of sodium citrate. In addition he is taking the routine exercises recommended by Buerger. Past experience indicates that the technical procedures used will assist in following accurately the progress of his case just as they aided in determining the diagnosis.

Studies of the peripheral circulation can be carried out without undue difficulty by methods that are well within the reach of every practitioner. No attempt has been made to discuss the more recent work that is being done on the capillary circulation, which at present is chiefly of physiological interest. On the other hand, in oscillometry and the histamine reaction two well recognized technical procedures are now available by which it is possible to obtain data of great practical importance about both the superficial and deep circulation of the extremities.

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NASAL SINUS DISEASE IN CHILDREN*

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Infections of the nasal sinuses in children are of greatest importance in pediatrics. In almost every instance in which infection of the tonsils plays an important part in systemic disease the condition of the nasal sinuses must be taken into consideration. If an infection is found in the nasal sinuses it is often of equal importance with the infection in the tonsils. It is the rule in children with heart disease or arthritis, when the removal of the tonsils has produced an improvement but the condition has not been satisfactorily controlled, that some infected lymphoid tissue has been overlooked, or there is an infection in the nasal sinuses, or both. A most important part of the treatment of these two conditions is the eradication of infection about the upper respiratory tract. It is axiomatic that this can be accomplished only by looking for infection in the nasal sinuses and if it is present, by eradicating it.

In every case of heart disease or arthritis in children that comes into our service for the removal of infected lymphoid tissue from the pharynx, the nasal sinuses are studied. After the tonsils have been removed we make a note on the chart to the effect that it is very difficult to remove all infection from the upper respiratory tract and that the patient should be returned to us if a satisfactory result has not been secured.

It is a common thing for the pediatrician to return these patients to us with the statement that following an acute upset of the respiratory tract, the systemic lesion has had an acute exacerbation, and there must be something left in the upper respiratory tract which is responsible for it. We make a second examination of the nose and throat, eradicate any infection that is found, and send the patient out with a clean throat and nasal sinuses. Because of the difficulty of stamping out nasal sinus infection in children completely, these patients are often again returned by the pediatrician for further treatment.

When we have a patient with nasal sinus disease and a heart or joint lesion, and this systemic condition pursues a satisfactory course after the first operative procedure on the nasal sinuses, we find that the nasal sinus condition is such that the sinuses remain clean after this first treatment. One can almost predict the course of the nasal sinus infection by a study of the systemic disease.

It is easy to eradicate an infection in faucial tonsils because the tonsils can be taken out and thrown away. An infection in the maxillary sinus is quite a different matter. The tooth buds in children are in the floor and the lateral wall of the sinus. There is no way of removing the sinus completely. The best we can do is to take out a portion of its nasal wall, clean the sinus, and secure ventilation and drainage. The walls that are left may be infected and only with difficulty and by prolonged treatment can the infection be satisfactorily controlled. In children without heart disease, arthritis or other systemic diseases deleteriously influenced by the infections, this is not a serious problem. With hygienic, dietetic and simple conservative treatment extended over months or years, a satisfactory result can be obtained. In cases, however, where there is need of haste in eradicating the sinus infection because of serious complications,

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it is exceedingly difficult to get a good result so far as the nasal sinus infection is concerned within a period of time which is satisfactory to the pediatrician. This is particularly difficult to accomplish in cases of children whose economic conditions do not permit a satisfactory home life.

It is difficult to make a good examination of the nasal sinuses in children without general anesthesia. The x-ray examination as a diagnostic procedure is not definite. In nephrosis, in allergic rhinitis, where there may be no infection of the sinuses, they may be completely blurred on the x-ray plate. The main reason for taking an x-ray picture of the sinuses in children is to determine whether or not a given sinus is present, and if it is present, to ascertain whether, on an anatomical basis, it is of sufficient size to have clinical significance. A blurred sinus on the x-ray plate, together with the presence of inflammation about the ostium of the sinus or in the pharynx, would suggest infection. Palpable glands in the neck are almost always present when there is infection of the nasal sinuses.

A most important procedure in diagnosing infection of the sinuses is a bacteriological and a cytological study of the nasal discharge taken from the ostium of the sinus. The presence of eosinophils in this discharge is very suggestive of an allergic rhinitis. An abundance of polymorphonuclear neutrophils indicates infection. It is rare to find an abundance of streptococci unless there is infection in the sinuses. The best way to make a diagnosis of infection of the sinuses in children is to combine a good clinical examination, including a laboratory study of the nasal discharge, with the examination by means of the roentgen ray. Certain things can be done with any child. The x-ray picture may be secured, the pharynx may be studied, the laboratory examination of the nasal discharge may be made, glands in the neck may be looked for, and the nose may be examined by anterior rhinoscopy. On the other hand, aspiration of the sinuses for bacteriological and cytological study, and the study of the interior of the nose with the nasopharyngoscope which can be done easily in the adult is impossible with young children. In many children it is necessary to remove the tonsils under general anesthesia. There is no reason why the examination of the sinuses should not be completed while the patient is under the influence of the anesthetic. It is easily done. In short, in every child in whom nasal sinus infection may have a definite bearing upon a co-existent systemic condition, if and when the tonsils are removed, a study of the nasal sinuses should be made at the time of operation in order that the pediatrician and the laryngologist may know exactly with what they have to deal.

The following diseases may be associated with nasal sinus disease:

Meningitis	Diarrhea in infants with dehydration
Brain abscess	Fever
Bronchiectasis	Cyclic vomiting
Lung abscess	Orbital and ocular infections
Sinus lung	Pyelitis
Bronchitis	Septicemia
Nephrosis	Allergic conditions
Nephritis	Chronic digestive disturbances
Malnutrition	Abdominal pains
Cervical adenitis	
Diphtheria carriers	
Head pains	

I do not mean to say that nasal sinus disease is necessarily the cause of any of these conditions. But the infection of these structures so often plays a prominent part in the clinical picture of these diseases that a complete diagnosis cannot be made without a study of the sinuses. Further, a satisfactory result cannot be secured except by attacking infection about the nose. In many instances this constitutes the main part of the treatment. In the so-called sinus lung and in some cases of bronchitis, little or nothing can be accomplished except by the eradication of a co-existent nasal sinus disease. When a diphtheria carrier has the organisms present in an infected sinus, they will persist until the infection of the sinus has disappeared. In allergic rhinitis with infection of the sinuses, particularly in cases where there is a history of repeated colds since early infancy, it is impossible to tell which was the primary lesion, the edematous swelling of the mucosa or the infection. A good result can be secured only by treating both conditions.

Anyone who thinks, because a patient with asthma has a chronic infection of a nasal sinus, that when the infection is eradicated the asthma will be benefited, is going to have many disappointments. The fact that an asthmatic has an infected sinus does not mean that there is any connection between the sinus disease and the asthma. On the other hand, in many cases of asthma where the pediatrician says that in spite of all his treatment, there has been no improvement in the asthma, it is advisable to take into consideration the significance of a chronic infection of a sinus if it is present.

Good results are often secured in children

who from infancy have had repeated attacks of nasal sinusitis and in whom such an attack often preceded the asthmatic seizure. To me it is necessary that the pediatrician should say that he has exhausted all of his resources before I attack infections in the sinuses. The results of sinus work in selected cases certainly justifies the operations that have been done.

In Still's disease, in bronchiectasis, and in other conditions, it is often difficult to evaluate the benefit derived from the treatment of the nasal sinuses. Nasal sinusitis is often responsible for malnutrition in children. Children suffering from malnutrition frequently are not benefited by the most meticulous dietetic treatment, but do get very satisfactory results from the eradication of infections about the upper respiratory tract. Many cases of Still's disease, bronchiectasis and similar conditions, suffering also from malnutrition, with the eradication of the chronic infection in the sinuses become so much better that the parents and others are inclined to feel that the bronchiectasis or the Still's disease have been favorably influenced, although in fact these conditions may be just as before. The improvement is due to a betterment of the general health. This has interfered with the evaluation of nasal sinus treatment, particularly in Still's disease.

Chronic infection of the nasal sinuses is often present in Still's disease. In this class of cases it seems to be particularly resistant to treatment. Cases of Still's disease in which we have been successful in eradicating the nasal sinus disease, have been benefited so far as the bouts of pain and fever and swelling of the joints are concerned. Treatment of the sinuses naturally has no influence whatsoever upon the deformity. After having seen many of these crippled children who suffered from repeated attacks of pain and swelling relieved of their acute exacerbations, I cannot help but feel that the work on the sinuses is justified.

The first step in the treatment of nasal sinus infection is to remove the cause. Infected lymphoid tissue must be removed from the nasopharynx. Lesions in the nose interfering with ventilation of the sinuses should be corrected. Allergic conditions must be controlled. Endocrine dysfunction is often an important factor. The hygiene of the home may need adjustment. Particularly are exercise and play in the sunlight indicated. Above all things, improper food habits must be controlled. It is surprising how many deleterious habits extending over many years will be found. The correction of these habits plus proper hygiene with an abundance of exercise often in itself gives excellent results when infected lymphoid tissue in the pharynx, if present, is removed. In cases uncomplicated by systemic disease this is usually all that is necessary. Operative work on the sinuses in children is not often indicated unless a systemic condition indicates haste in the eradication of the infection.

When you have a serious complication of nasal sinusitis, as for instance, a hemorrhagic nephritis, or a heart lesion, and you have reached the stage of the systemic disease when the nasal sinuses should be operated upon, if operation is to be done, the exigencies of the situation demand haste in the eradication of the nasal sinus disease and the conservative treatment just outlined is not advisable.

In children under ten years of age, only occasionally do we need to consider the sphenoidal or the frontal sinuses. The sphenoid is usually treated as a posterior ethmoidal cell.

The canine fossa operation often done on the maxillary sinus in adults is contraindicated in children with a very rare exception when the outer wall of the maxillary sinus is completely necrotic. In such instances the face is swollen, and usually there are discharging sinuses with sequestration.

Not in more than one out of 100 cases of children with chronic nasal sinus disease with systemic complications is it necessary surgically to attack the ethmoidal labyrinth. This labyrinth should never be attacked unless one is convinced that osteomyelitis is present and that there is a definite connection between the infection in the ethmoid cells and the systemic disease. Further, one must be convinced in advance that the eradication of the ethmoid disease will bring about an improvement in the systemic condition sufficient to justify the operation.

In 99 per cent of children with chronic nasal sinus infection and systemic complications, the surgical procedures in the nose are limited to the removal of obstructing lesions and a very small meatal window in the nasal wall of the maxillary sinus for ventilation and drainage. This, combined with cleaning the nose with suction, the occasional cleansing of the maxillary sinus, together with the use of some simple antiseptic in the nose, should meet the situation.

Usually these patients are greatly improved following the first operative procedure. The

nose becomes clean and apparently well. Almost always there is a return of the trouble in the nasal sinuses. Each time they should be carefully studied and such work done on the nose and throat as is indicated. Throughout the treatment it is necessary to get rid of any recurring infected lymphoid tissue in the pharynx. It is particularly important to watch the lingual tonsil and if it is enlarged and manifestly infected, to remove it.

DISCUSSION (*Abstract*)

Dr. H. Marshall Taylor, Jacksonville, Fla.—Dr. Dean has shown us the proper method of securing nasal cultures. There is one phase of this subject which, so far as I know, he has not covered, and that is the investigation of the aspergillus or the mycotic infections of the sinuses. This is work that has been largely ignored and until the proper culture media is used as a routine, we shall not know just how many sinus infections are due to aspergillus. Aspergilli, as a rule, are saprophytes, but they may become parasites and secrete true toxins. This specimen is an *Aspergillus fumigatus* which was cultured from a patient of mine who had a chronic maxillary sinusitis. The secretion from this sinus was planted on a culture medium containing:*

Maltose	12.75 gm.
Malt extract	15.00 gm.
Dextrin	2.75 gm.
Glycerin, C. P.	2.35 gm.
K_2HPO_4CP	1.00 gm.
NH_4Cl C. P.	1.00 gm.
Bacto-peptone	0.78 gm.
Bacto-agar	15.00 gm.

Dissolve 50.63 grams in 1,000 c. c. of distilled water by boiling. The resulting complete medium is then ready to dispense into containers for final sterilization. The final pH is 5.0. Slants are used in large test tubes or plates are poured. Make a heavy inoculation of the suspected material. Incubate at least two weeks. A temperature of 37.5° C. is optimum, but it may keep at room temperature.

A growth may be obtained in eighteen to twenty-four hours, but the average period is about seven days. The characteristic brown, black, or green color of the aspergilli requires several days to develop.

Aspergilli are frequently found in cultures made from old lung abscesses. Until all of these cases are cultured for aspergillus, almost as a routine, we cannot determine how frequently aspergillus infections of the sinuses exist.

Dr. J. N. McLane, Pensacola, Fla.—What operative procedure would Dr. Dean use in treating a child of eight years of age, who has a polypoid degeneration of the antra? I have under my care a child of this age, who has definite polyps in her antra as shown by radiographic study of the sinuses after lipiodol injection of the antra. I have hesitated about doing a Caldwell-Luc operation, as the facial bones are not fully developed.

Dr. Dean (closing).—In children with nasal sinus disease without systemic or local complications, surgical procedures are usually contraindicated. Attacking the cause is the essential part of the treatment. In heart lesions in children, in arthritis, in sinus lung, and in many other conditions the essential part of the treatment is the eradication of infection about the nose and throat.

*Bacto Wort Agar (dehydrated). for cultivation of yeasts. Digestive Ferments Co., Detroit, Michigan.

SYMPATHETIC OPHTHALMIA*

REPORT OF TWENTY-EIGHT CASES

By LAWRENCE T. POST, M.D., F.A.C.S.,
St. Louis, Missouri

The material in this paper is based upon the study of twenty-eight cases of sympathetic ophthalmia, in all of which the first affected eye was examined pathologically and in one of which the sympathizing eye also was examined. Of these patients ten came under my personal observation. The pathological examinations were all made by Dr. Harvey D. Lamb in the Department of Ophthalmology, Washington University. Several of these cases have been published individually. The total number occurred in slightly over a thousand specimens. A few of them were so unusual as to justify confirmation of the diagnosis by other pathologists so that these have been examined by such authorities as the late Dr. Ernst Fuchs and Dr. F. H. Verhoeff.

No attempt has been made to review the vast literature of the subject of sympathetic ophthalmia, but merely to present this series in a somewhat statistical manner, referring especially to a few of the cases which seem of unusual interest. The writer was stimulated to make this study by the splendid report of Dr. Bernard Samuels on "Significance of Specific Infiltration at the Site of Injury in Sympathetic Ophthalmia" presented before the American Ophthalmological Society in 1932, and a later paper on scleral involvement in sympathetic ophthalmia read by Dr. Samuels before the same Society in 1933 and published in the *Archives of Ophthalmology* for August, 1933. These papers will be referred to frequently in this report.

Any series of twenty-eight cases of sympathetic ophthalmia permits of presentation from

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*From the Department of Ophthalmology, Washington University, St. Louis, Missouri.

many angles and these cases will undoubtedly be reported later in connection with numerous individual points. The clinical aspect was chosen because it is in that phase of the subject that the writer is especially interested and, furthermore, he recognizes that he is not qualified as an expert pathologist and has accepted the diagnoses of Dr. Lamb and his pathological interpretations. The detailed study of the sections will probably be more valuable than the clinical aspect and will be given at another time by Dr. Lamb.

In the opening paragraph of Dr. Samuels' paper on "Specific Infiltration at Site of Injury, etc.," the author refers to the first scientific description of sympathetic ophthalmia which was given by Wm. Mackenzie in 1835, and the advocacy of Augustin Pichard, in 1851, of the excision of the injured eye as a means of preventing inflammation in the fellow eye. The third and final advance of great significance (again quoting Samuels),

> . . . "was the demonstration by Ernst Fuchs in 1905 from a painstaking study of anatomic preparations that sympathetic ophthalmia is a disease entity . . ."

From Dr. Samuels' examination of one hundred and one cases he concluded that a perforation of the globe was necessary to the production of sympathetic ophthalmia and that the primary focus was the wound. In his series two cases showed the disease in the wound only. None in my smaller series was similar to those two in that respect, but very many of them did show sympathetic disease in the wound. Against his thesis are the undoubted cases of sympathetic disease in which no penetration of the eyeball has taken place. Dr. Samuels mentions these briefly in referring to the few cases of sympathetic disease associated with degenerating sarcomata, but dismisses them without attempting explanation. In my series there were two cases of this sort: in *Case 1,* a moderately severe blow with an unpointed stick on a normal eye, clinically and pathologically resulted in no indication whatsoever of penetrating injury (though serial sections were not made). In *Case 3,* the condition developed without injury in an eye containing a necrotic sarcoma. The pathological diagnosis in the latter case was confirmed by Dr. Verhoeff. Such histories as these would seem definitely to exclude the theory of the necessity of exogenous wound infection as a primary cause, though not eliminating the possibility of the localization or activating of organisms in the eye, which were already present in the body or on the mucous membrane. The traumatic case might theoretically be explained by corneal penetration without penetrating injury, such as Calmette accomplished with tubercle bacilli. Whenever the question arises as to whether there was a penetrating injury in an eyeball someone always inquires whether serial sections were made. Though it is possible theoretically to overlook penetration *in vivo* and *in vitro* even when they are carefully sought, I should like to inquire from the pathologists present whether they have ever encountered in sections a penetrating wound which was not discovered by the clinician or by themselves in the gross specimen. To my mind a careful examination of the globe in case of a blow from a stick could scarcely fail to reveal a penetrating injury if one were present and such an examination is sufficiently exclusive. That such a blow might break down the conjunctival defense and so permit the introduction of organisms having an affinity for ocular tissue is more reasonable, but even this assumption does not explain the fairly numerous cases of undoubted sympathetic disease associated with degenerating sarcomata.

In addition to the hypothesis of exogenous infection, an important contribution to the possible etiology of sympathetic ophthalmia was the suggestion that the inflammation might be allergic, due to liberated uveal pigment. The principle advocate of this hypothesis and worker in this field is Dr. Alan Woods.

In discussion of Dr. Samuels' paper, Dr. Jonas Friedenwald said he thought that if the disease were due to allergy to uveal pigment we should find uveal pigment in the specific lesions. In a series of twenty cases he found ten in which small, tubercle-like lesions in the retina contained pigment granules. He thought, furthermore, that if one injected uveal pigment in patients at other sites one should produce an epithelioid cell reaction similar to that in sympathetic ophthalmia. He said that

> "in two patients with sympathetic ophthalmia, however, the character of the reaction about the skin test was identical with that seen in the eye, great masses of epithelioid cells extending two or three millimeters from the site of pigment deposit."

In normal persons there was very slight reaction at the site of injection and though many lymphocytes were seen no epithelioid cells developed. In ten years of testing patients with uveal pigment Woods has found only one patient who had no uveitis but who clinically had a positive skin reaction to uveal pigment. The

one patient would, however, suggest, according to Friedenwald, that allergy to uveal pigment was not alone sufficient to cause the disease.

Only one patient in this series was tested with uveal pigment, but I believe that this should be done wherever possible to help clear up the mystery of this disease. The sections have not been studied to determine the presence of uveal pigment in the specific lesions.

Dr. Samuels reported fairly frequent involvement of the sclera in his cases. In only a few of our cases was the sclera involved in sections which were examined. This, of course, does not exclude the possibility of the presence of this feature in other sections of the same eye. This finding seems to be associated with advanced ocular involvement.

Certain tables from our material are of interest.

Table 1

CAUSE OF SYMPATHETIC INFLAMMATION

Penetrating injuries	18 or 64.3 per cent
No penetration of globe	2 or 7.1 per cent
Operative wounds	8 or 28.6 per cent
Total	28 or 100 per cent

The attention is attracted by two features: first, the large percentage of cases due to operative wounds, namely, 8 cases, or 28.6 per cent; and second, the two cases, or 7.1 per cent, in which no penetration of the globe occurred.

Table 2

INVOLVEMENT OF FELLOW EYE

Fellow eye involved	25 or 89.3 per cent
Uninvolved	3 or 10.7 per cent
Total	28 or 100 per cent

Table 2 indicates that only 3 of 28 eyes were enucleated early enough to prevent involvement of the fellow eye.

Table 3

OUTCOME IN CASES IN WHICH FELLOW EYE WAS INVOLVED

Restoration to normal vision	13 or 52 per cent
No restoration to normal vision	11 or 44 per cent
Unknown	1 or 4 per cent
Total	25 or 100 per cent

Table 3 is, I believe, unusual in the number of favorable cases, representing 54.2 per cent of 24 cases in which the second eye was involved and the outcome known.

Table 4

RELATION OF OUTCOME TO CAUSE WHEN FELLOW EYE WAS INVOLVED

Of the operative cases, 3, or 50 per cent, did well
Of the operative cases, 3, or 50 per cent, did badly
Of the injury cases, 10, or 55.6 per cent, did well
Of the injury cases, 7, or 38.9 per cent, did badly
Of the injury cases, 1, or 5.5 per cent, unknown

This table suggests that there is little difference in the outcome whether the disease is caused by injuries or by operative wounds.

Table 5

SYSTEMIC TREATMENT IN CASES WHICH RECOVERED

Arsphenamine and salicylates	2
Salicylates and mercury	2
Salicylates and typhoid-paratyphoid vaccine intravenously	2
Typhoid-paratyphoid intravenously	2
Salicylates	1
Autohemotherapy, milk injections, salicylates and uveal pigment	1
Removal of focal infection	2
Unknown but extensive	1
Total	13

Salicylates used in 8 cases
Typhoid-paratyphoid in 4 cases
Mercury in 2 cases

This table is only slightly suggestive because similar treatments were used in unsuccessful cases. Its only value may be an indication of the kind of treatments used in a series of cases which shows a good percentage of recoveries. A matter of some significance was that all cases received a great deal of systemic medication.

In this series there were some especially interesting cases which are here presented. *Case 3* will be dealt with later in a separate paper and will be given here in outline only, as a clinical rather than pathological presentation.

Case 1.—Lucille G., aged 6 years, was struck in the right eye with a blunt stick. The patient was seen within two hours after the injury by my associate, Dr. William E. Shahan. On the following day there was much hemorrhage in the anterior chamber and increased intra-ocular tension. Very careful examination showed several small superficial abrasions on the cornea, but no other wound. The hemorrhage was gradually absorbed and the eye became quiet. Sixty-nine days later the vision was light perception in the injured eye and there was beginning sympathetic ophthalmia in the fellow eye. Enucleation was done on this date. The enucleated eyeball was examined with great care for any possible penetrating injury, but none was found. Serial sections, however, were not made. The pathologic specimen showed typical sympathetic disease in the iris and ciliary body. There was also edema of the nerve head.

Case 2.—Sarah J., aged 52 years, in the left eye showed severe chronic iridocyclitis; many brown spots of keratitis punctata; many rounded nodules in the iris stroma of color similar to the iris, though slightly more gray. Throughout the iris in all regions there were posterior synechiae, and there was gray exudate in the pupillary space. The Wassermann in the blood was negative. A diagnosis of chronic uveitis was made. Treatment for many months was without avail. There was much pain with hypertension, for which several corneal paracenteses were done. Finally the eye was enucleated July 23, 1931, as vision was reduced to poor light perception and the eye was still painful.

Up to August 31, 1931, vision in the second, that is, the right eye, was 6/5, at which time vision was 6/20. A uveitis developed and gradually became worse until January, 1932, at which time vision was 1/60; uveitis was severe with keratitis punctata. The patient was hospitalized and treated. Vision increased to 1/12 under treatment for tuberculosis, for which the condition was taken as the pathological specimen of the first eye had suggested it. The right eye did very badly, became unbearably painful and was enucleated November 5, 1932. No operative procedure had ever been done on this eye.

The sections from this eye showed, not chronic inflammation, but sympathetic ophthalmia.

Case 3.—In Wm. S., aged 38 years, the right eye became inflamed in October, 1925. During the following two months this eye was treated varyingly. Tension was up and down. A diagnosis of probable hemorrhagic glaucoma was made. Vision was very poor. At this time the fellow eye showed a uveitis. Enucleation of the right eye was performed. A large necrotic melanosarcoma was found and many areas of sympathetic disease. This case was reported by Dr. F. O. Schwartz in the *American Journal of Ophthalmology* in January, 1927.

Case 4.—This patient had a penetrating injury of the globe on July 27, 1926. Panophthalmitis occurred on August 11, and evisceration was done on that day. On August 28, thirty-one days after the injury, uveitis was noted in the fellow eye. Sections showed sympathetic infiltration in inflammatory exudate in the extreme posterior part of the eye. Possibly this infiltration came from the posterior emissary vessels and nerves after the evisceration. The case was reported by Dr. Shahan in the *American Journal of Ophthalmology* in February, 1927.

It is possible that the sympathetic process occurred after the evisceration, in which case the failure of this proceeding to check sympathetic inflammation is noteworthy, or it is possible, and more likely, that the sympathetic process had occurred prior to the evisceration and had been present in the fellow eye or else in the general system without producing visible evidence for seventeen days.

Case 5.—Miss Clara M., aged 37 years, suffered a penetrating injury from flying glass. Four months later uveitis began in the fellow eye. Enucleation of the injured eye was done at once, but vision in the remaining eye failed to 16/240. It gradually recovered to 16/75. One year after enucleation of the first eye a mature cataract was present in the second eye and the eye was quiet. What was intended to be a preliminary iridectomy was performed without complications, but the eye never recovered from a uveitis which sprang up, ultimately destroying the eye.

CONCLUSIONS

No definite conclusions can be drawn from the material here given, but these points are suggested: (1) a penetrating injury is not essential to the development of sympathetic ophthalmia; (2) two cases indicate that the attention to foci of infection is important in treatment of the sympathizing eye; (3) one case suggests the inadvisability of operating early on sympathizing eyes; (4) in the one case in which the sympathizing eye was examined, the finding of previous observers that the picture in the fellow eye is similar to that in the exciting eye, does not necessarily hold, though the appearance in the sympathizing eye was similar to that of sympathetic ophthalmia in general; (5) this same case indicates either that a severe chronic inflammation in one eye can cause sympathetic disease in the fellow eye, or that in such a chronically inflamed eye a very small penetrating injury of the cornea only, such as paracentesis, is sufficient to excite the disease in the fellow eye; as several cases in this series occurred in the fellow eye many months and even years, one being nineteen years, after the original injury, the removal of a sightless, injured eye should always be urged, as it ever remains a potential source of sympathetic ophthalmia.

DISCUSSION (*Abstract*)

Dr. Karl S. Blackwell, Richmond, Va.—To the average ophthalmologist, these cases come infrequently; and when they do come, they bring with them such dire consequences that his attention is fixed more upon the best method of saving the sight than upon a scientific study of the pathological processes which are taking place in the eyes.

In the study of Dr. Post's series one cannot fail to be struck with the large percentage of cases following operation. The fact that more than half recovered is truly gratifying and reassuring.

The two very interesting cases in which there was no penetration of the ball are of great value in adding weight to the much discussed question of whether or not one can have sympathetic ophthalmia without a penetration of the ball.

His *Table 4* on the relation of the outcome to the cause when the fellow eye was involved is significant, as we have been led to feel that severe wounds in the ciliary region were the ones to be watched most carefully. Parsons thinks that this region is the danger

zone only on account of the incarceration cyclitis which prevents an easy process of cicatrization.

Case 5 shows that there are often latent and unknown factors which have to be taken into consideration in operating upon an eye in which there have been symptoms of sympathetic ophthalmia.

Elschnig thought that an injury to the exciting eye caused an absorption of the uveal pigment and this produced a hypersensitiveness of the whole body, and especially of the uvea of the other eye.

Dr. Alan Woods has done much work along this line. He used uveal pigment as an antigen and found patients with sympathetic ophthalmia hypersensitive to the pigment when it was injected intracutaneously. It is thought that pigment allergy impairs normal resistance and allows some unknown agent to act.

Dr. Bernard Samuels, who has done outstanding work with sympathetic ophthalmia, reported before the American Ophthalmological Society ninety-four cases in which there was scleral involvement in 70 per cent. He feels that the breaking down of the scleral tissues plays an important part in the severity of certain cases of sympathetic ophthalmia.

The trouble is in the posterior zone near the macular region or optic nerve. As he feels the possibility that a specific infiltration may occur outside of the eye posteriorly, he suggests that a long piece of the optic nerve and orbital tissue be removed at the time of the enucleation and that radium be applied to the orbit.

Dr. Dunbar Roy, Atlanta, Ga.—For many years I believed that so-called sympathetic ophthalmitis was a myth, but when I saw five cases in two years, then I realized that it was a distinct entity.

Twenty-six years ago I read a paper before the American Academy of Ophthalmology and Otolaryngology in which I gave my experience with this pathologic condition. I called attention to the histo-pathologic findings of Fuchs in these cases. However, this added nothing to the best method of treatment. The condition is still obscure and also its etiology. Many questions arise in the course of the management of these cases, and I believe that experience is the best teacher. However, such cases are becoming less and less frequent, so that there must be something in our aseptic surgery.

Dr. Post (closing).—In the series here reported the time of involvement of the second eye varied from three weeks to nineteen years after injury or operation.

A case from the literature was mentioned in the discussion, of sympathetic disease in the second eye, forty-eight hours after injury to the first. If authenticated, this would be almost conclusive evidence against bacterial etiology.

If the injured eye is better than the sympathizing eye when the patient is first seen, it is advisable to retain both eyes, as the injured eye may prove the better in the end.

I do not believe it is possible to make a positive clinical diagnosis of sympathetic ophthalmia in an injured eye nor can one differentiate clinically sympathizing uveitis from other plastic uveal inflammations; but the occurrence of uveitis in one eye shortly after a penetrating injury to the other eye is tremendously suggestive. The diagnostic value of uveal pigment tests is seemingly very important, the difficulty being the obtaining of suitable pigment on short notice.

Apparently children are more susceptible to sympathetic disease than adults. In my series the percentage of children was high, but I do not have data here to show the incidence of penetrating injuries in adults and in children which would be necessary to estimate the relative frequency of sympathetic inflammation at different ages.

My studies do not suggest any new hypothesis concerning the etiology of sympathetic ophthalmia.

PARALYSIS OF THE DIAPHRAGM AS A THERAPEUTIC AGENT*

By RUSSELL B. BAILEY, M.D.,
Wheeling, West Virginia

In 1925, John Alexander[1] published his book, "Surgery of Pulmonary Tuberculosis." During the same year he published an article in the *Journal of Outdoor Life* outlining, for the layman, the therapeutic advantages of surgery in tuberculosis. Prior to this time only a few clinics in North America had done a great deal in this field. Following the increased interest stimulated by Alexander's contributions in the treatment of tuberculosis, the procedures outlined were generally applied to other diseases of the lungs and pleura.

The most popular and frequently used procedures were operations devised to produce paralysis of the diaphragm: phrenicotomy, phrenectomy or phrenic exeresis, and the crushing and freezing of the phrenic nerve.

Sturtz[2] was the first to recommend section of the phrenic nerve for the treatment of pulmonary cavities. It was soon found, however, that simple section of the phrenic nerve did not produce complete paralysis of the diaphragm. To Felix and Goetze[3] we owe the first careful anatomical discussion of the nerve supply of the diaphragm. Their contributions described the various anatomical distributions of the phrenic nerve.

It has been variously estimated that, following diaphragmatic paralysis, the volume of the thorax is reduced from 200 to 500 c. c. The rise of the diaphragm is due to the negative intrathoracic and the positive intra-abdominal

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pressure. The degree of elevation of the diaphragm is determined by the retractability of the lung and by the presence or absence of adhesions between the diaphragm and parietal pleura. As the diaphragm recedes into the thorax, the underlying lung is relaxed. The more elastic portions of the lung, that is, the normal and unfibrosed areas, will be the first to take up the slack, whether they be at the base, the middle or the apex of the lung. Somewhat later on, the cavity walls collapse and the fibrous areas shrink, so that, as an end result, the normal lung tissue expands to fill the thoracic cavity. The medial aspect of the diaphragm is held firmly by the central tendon, while the mid and lateral portions are muscular. The diaphragm, therefore, rises higher from the costophrenic angle to the mid-portion than from the mid-portion to the central tendon. The direct compression is upward and inward, and as the size of the thoracic cavity is gradually reduced, there is a corresponding decrease in the size of the lung.

The immobilization and rest of the diseased tissue increases the rapidity of healing by promoting the lymphatic drainage and reducing the amount of exudate. Opinions differ as to whether cough and expectoration are made easier. Carlson, Balland, Wilson and Graham[4] reported a very instructive study, based upon the elimination of iodized oil and foreign bodies from the lungs. From their experiments, they could not accept the frequent statement that paralysis of a leaf of the diaphragm facilitates cough and expectoration. On the contrary, their experiments proved that the elimination of iodized oil and foreign bodies from the lungs was made more difficult on the paralyzed side. The fact remains, however, that from a clinical standpoint, expectoration and cough are improved and made easier following diaphragmatic paralysis.

INDICATIONS FOR HEMI-DIAPHRAGMATIC PARALYSIS

Hemi-diaphragmatic paralysis is of a great deal of value in treating certain forms of pulmonary tuberculosis. In a general way, one may say that hemi-diaphragmatic paralysis should be considered in any case of unilateral pulmonary tuberculosis, with or without cavitation, after a reasonable period of observation. There is an abundance of literature outlining the indications for phrenicotomy in this disease. A recent article in the *Journal of Thoracic Surgery,* by Alexander,[5] is a most comprehensive and instructive study. The follow-up reports demonstrate, beyond any doubt, the efficiency of unilateral diaphragmatic paralysis in selected patients with pulmonary tuberculosis.

Intrapulmonary cavities, whether in the base, the middle or the upper lobes, are frequently influenced and closed as the result of the reduction in the size of the thorax from the diaphragmatic paralysis. Occasionally one sees a lobe of the lung shrink until it cannot be demonstrated. Wolf and Lossen[6] have reported such a case, the interlobar fissure rising to the point where it became fused with the upper mediastinum.

Bronchiectasis is a disabling and difficult disease to treat. The unilateral forms, which are most frequently due to foreign bodies, unresolved pneumonia, or low-grade suppurative pneumonitis, will frequently respond to the compression made by paralysis of the diaphragm. There are a great many reports of small series, with varying results. A large percentage of the unilateral variety are either cured or improved by paralysis of the diaphragm on the involved side.

Long-standing pulmonary disease occasionally produces distortion of the great vessels in the upper mediastinum, tension on the vagus nerve, trachea and esophagus. This group of patients frequently has annoying pain and there may be attacks of cardiac insufficiency upon exertion, tachycardia and attacks of syncope. All of these symptoms may be relieved by a reduction in the size of the thorax by diaphragmatic paralysis.

In the treatment of large pleural cavities, as a result of poorly treated or neglected empyema, temporary or permanent paralysis of the diaphragm in conjunction with decortication of the lung will frequently obviate the necessity of extensive rib resection, such as the Estlander or Schede operations.

Following lobectomy, elevation of the diaphragm will reduce the size of the thorax, relieve the tension on the amputated stump, and eliminate the emphysema, which may follow in the remaining lobes.

In transpleural mediastinotomy for mediastinal or intrapulmonary tumors, temporary paralysis, by squeezing or cocainizing the nerve, will eliminate the churning movement of the diaphragm and facilitate the operative procedure.

Temporary diaphragmatic paralysis is of considerable aid in operations upon the diaphragm

itself, whether it be an acute rupture or an acquired or congenital hernia. It increases the ease of operation and relieves a certain amount of tension, which, in turn, is an aid to healing.

Temporary diaphragmatic paralysis in the treatment of intractable hiccoughs has received some attention and favorable results have been reported. Angle[7] reports a case of bilateral phrenicotomy for persistent hiccoughs with a good result. No disability followed. In the Wheeling Clinic we have used this procedure on one occasion. The left phrenic nerve was exposed in the neck and lightly squeezed. The left diaphragm was immediately paralyzed. The severity of the hiccoughs was decreased, yet the patient continued to hiccough at intervals for three days following the operation. Examination of the patient three months later revealed normal excursion of the left diaphragm.

CONTRAINDICATIONS

There are certain forms of pulmonary suppuration, such as the markedly sacculated forms of bronchiectasis or suppurating disease following stricture of a bronchus, in which paralysis of the diaphragm is contraindicated. In this type of lesion there is danger of obstructing drainage of the affected areas, producing abscess or gangrene. Long standing, chronic, multiple abscesses in the basal lobe, with extensive fibrosis, will not respond to elevation of the diaphragm. Extensive disease in the contralateral lung is a contraindication, except where bilateral phrenicotomy, pneumothorax or some of the allied procedures of thoracoplasty are contemplated, and then the keenest judgment must be used.

BILATERAL DIAPHRAGMATIC PARALYSIS

Bilateral diaphragmatic paralysis as a therapeutic measure is not frequently referred to in the American literature. Lemon[8] reported his experimental work on dogs, in which he found that bilateral paralysis of the diaphragm did not interfere with the normal activity of the dog. The endurance of the dog with bilateral phrenic paralysis compared favorably with that of the normal dog.

Until recent years the diaphragm was considered the essential respiratory muscle. The physiologist's present conception, however, is that the diaphragm plays only a minor role in respiration and acts more as a division between the abdomen and thorax. The principal function of the diaphragm is to aid coughing, sneezing and phonation. They are the only acts which require complete play of the diaphragm.

Marc Iselin[9] refers to cases of tetany of the diaphragm during the World War which were saved by bilateral diaphragmatic paralysis. Following the paralysis, artificial respiration was resorted to, and a few patients recovered after a long period of time.

Iselin reports a case of Sauerbruch's, an infant with bilateral pulmonary tuberculosis, in which a bilateral phrenic exeresis was done. An arrest of the tuberculosis followed and after three and a half years the diaphragm was high and immobile. The child was perfectly normal and could carry on and do the usual athletic exercises prescribed for normal children.

In children the thorax is flexible. This makes it compensate much better than the adult thorax. Many of the European authors feel that the greater influence of diaphragmatic paralysis is on the basal lobes.

Curti[10] refers to 132 cases in the literature and adds a case of his own. This case had a bilateral phrenectomy for bilateral tuberculosis. After a period of time the patient was able to return to work.

In another article Curti[11] discusses the importance of bilateral phrenicotomy and gives, as the first indication, a diffuse fibrous type of tuberculosis, with the greater portion of the disease located in the basal lobes. He reports five cases in detail and states that all the patients had a sense of well being immediately after the operation. There was a decrease in temperature. Cough and expectoration were partially relieved. He reports no circulatory disturbance and no dyspnea of pulmonary origin but, on the contrary, a feeling of relief manifested by greater energy and better appetite. The results of the operation after two years were as follows: one patient died ten days postoperatively with cardiac collapse. Another died three months after operation with tuberculous enteritis, but the pulmonary symptoms were improved. In one case there was no beneficial result as far as the pulmonary lesion was concerned, yet the patient's general health was improved. The two other patients had, as he expressed it, gratifying relief from the treatment. In his conclusion he advised the use of bilateral phrenicotomy in the same way that bilateral pneumothorax is employed.

At the Wheeling Clinic, we were tempted to use a bilateral phrenicotomy on two occasions. The first was in a case of bilateral pulmonary tuberculosis with large cavities. Following paralysis of the right diaphragm, the patient im-

proved and the cavity closed. She still had the large cavity on the left side. Phrenicotomy was done on the left side. The cavity eventually closed, but subsequently opened. There was a marked reduction in the vital capacity, due to the extensive pulmonary disease and reduction of lung tissue so that there was, upon the least exertion of the patient, extreme dyspnea. The patient died some eight months after the second operation.

The second was a case of bilateral bronchiectasis, with large amounts of sputum. Following phrenicotomy on the right side, which showed the greatest involvement, the patient was partially relieved. Some months later, at the insistence of the patient, a phrenicotomy was done on the left side. Within a half hour following the second operation the patient was unable to raise sputum and almost drowned in her own secretion. After this it was necessary to keep her in an extreme Trendelenburg posture, so that she could eliminate the collected sputum. The patient died within three months after the second operation.

It would seem, from the data available, that bilateral diaphragmatic paralysis in intrapulmonary disease has an extremely limited field. From our experience and the results of the experiments of Carlson,[4] *et al.*, the lungs must be relatively dry, as the elimination of sputum is made very difficult. The operation is more applicable to children than adults because of the elasticity and compensability of the child's thorax. There must be a reasonable certainty that the vital capacity will not be reduced to such a degree that the patient cannot carry on, even though the disease should be arrested.

It is our opinion that bilateral diaphragmatic paralysis has a very limited field and should be undertaken only after careful consideration of all the facts at hand. There are many other therapeutic combinations, which are more rational and less dangerous, yet there is an occasional case in which bilateral diaphragmatic paralysis is of value.

THE OPERATION

There is nothing very difficult about operations upon the phrenic nerve, if one has a fair knowledge of the anatomy in the neck and keeps in mind the frequent variations of the phrenic nerve. The operative technic has been well described by Alexander,[12] and recently Decker[13] has outlined another method of approach suggested by Lilienthal.

There are three common anatomical variations of the phrenic nerve in the neck. In each instance the principal fibers which constitute the phrenic nerve arise from the third, fourth and fifth cervical trunks. The distribution most frequently found is that in which the fibers fuse high in the neck and the phrenic nerve thus formed passes obliquely across the scalenus anticus muscle into the thorax. In the second type of distribution, the nerve fibers fuse high in the neck and the phrenic nerve then passes distinctly outward, sometimes being imbedded in the muscle itself. It is occasionally joined by a small branch from the brachial plexus, as it enters the thoracic cavity. In the third type, the nerve fibers pass obliquely downward, across the anterior scalenus muscle, fuse low in the neck, and the main trunk is then joined by a typical subclavian accessory root. If the operator will bear in mind these anatomical variations, very little difficulty will be encountered in locating the nerve supply to the diaphragm.

Where permanent paralysis of the diaphragm is desired, either exeresis or a resection of two inches of the main trunk of the phrenic nerve will usually accomplish the desired result. These procedures are simpler and easier to perform than the radical phrenectomy described by Goetze.[14]

A great many complications have been reported, many of which are due to improper operative technic and the inability of the operator to recognize the phrenic nerve. There are two serious but unavoidable accidents which should be mentioned. Hemorrhage into the mediastinum may occur when the nerve is evulsed from the thorax, due to the injury of a blood vessel lying adjacent to the nerve. Several cases of Horner's syndrome have been reported, many of which were only temporary. In our series of two hundred twenty-five cases, this phenomenon occurred on four occasions. In only one of the four cases was the syndrome permanent. The percentage of serious complications is very small, so small, in fact, that one is justified in eliminating the consideration of these unfavorable possibilities when discussing the operation with a patient.

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DISCUSSION (*Abstract*)

Dr. I. A. Bigger, Richmond, Va.—Dr. Bailey says that paralysis of the hemi diaphragm should be considered in any case of unilateral pulmonary tuberculosis, with or without cavitation, after a reasonable period of observation. I quite agree with this statement and am inclined to go even further and say that paralysis of the hemi-diaphragm should be considered, and I believe used, in a large percentage of cases of early unilateral pulmonary tuberculosis as soon as the diagnosis is made. It seems to me that the situation is much the same as that which confronts us in acute appendicitis, except that there is of course less of the emergency element. We all know that a very considerable percentage of cases of acute appendicitis will clear up under conservative treatment, but few surgeons advise such treatment. This is right and proper, because it is less dangerous to do a clean appendectomy than to take the chance of having the patient develop a ruptured appendix. Similarly, it is impossible to say with any degree of accuracy what progress will be made under conservative treatment in any given case of early unilateral tuberculosis. Since temporary paralysis of the diaphragm is not a serious operative procedure and does not produce permanent disability, but does give considerable relaxation and added rest to the diseased lung for several months, I believe it should be used more frequently early in the disease. In other words, I consider temporary paralysis of the diaphragm less serious than pneumothorax. Permanent paralysis, on the other hand, should be reserved for the more advanced cases with cavitation or extensive fibrosis.

In addition to the indications for phrenic paralysis in pulmonary tuberculosis as suggested by Dr. Bailey, I would like to add that I believe the diaphragm should be permanently paralyzed before the lung is allowed to expand in any case of extensive cavitation which is treated by pneumothorax.

In my experience, phrenic paralysis has frequently given unsatisfactory results when used in the treatment of suppurative conditions in the lung. There has often been litt'e or no response. In two cases of basal bronchiectasis, elevation of the diaphragm produced obstruction in the dilated bronchi with serious consequences.

I have found temporary paralysis of the diaphragm to be of great value in hastening the obliteration of large lung abscess cavities, especially after surgical drainage had been established. I have also found temporary paralysis of the diaphragm of value when this structure was to be used to repair defects in the chest wall resulting from severe trauma or from the excision of large malignant tumors of the chest wall.

It seems to me that Lemon's work on bilateral phrenic paralysis in dogs is not applicable in clinical work because the surface area of the diaphragm as compared with the surface area of the chest wall is considerably less in dogs than in human beings, which would seem to indicate that the diaphragm is of less value as a respiratory muscle in dogs. Also, in cases of pulmonary disease in which bilateral phrenic paralysis might be indicated, the vital capacity is already greatly reduced, so that the added reduction from complete paralysis of the diaphragm would of necessity cause respiratory embarrassment. I believe, therefore, that permanent bilateral phrenic paralysis is very rarely indicated.

To summarize, I believe that the best results from phrenic paralysis are obtained in cases of pulmonary tuberculosis. I also believe that the results wou'd be even better if the procedure were used more frequently early in the disease. Permanent bilateral paralysis of the diaphragm is very rarely justifiable, but temporary paralysis of the entire diaphragm may be indicated in persistent hiccough.

Dr. Bailey (closing).—I purposely omitted a detailed discussion of the indications for operations on the phrenic nerve, in the treatment of pulmonary tuberculosis. I am sure we could all spend most of the afternoon discussing this particular subject.

The point which I particularly wanted to emphasize was that operations on the phrenic nerve are of considerable value in the treatment of carefully se'ected cases of intrathoracic disease.

The operation does not necessarily require the skill of a specialized thoracic surgeon, but may be done by any general surgeon, who is familiar with the anatomy of the neck.

THE TRAINING DESIRABLE IN FUTURE PRACTITIONERS OF MEDICINE IN THE PROMOTION OF CHILD HEALTH*

By J. H. MASON KNOX, JR., PH.D., M.D.,
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An adequate child hygiene program in a community should provide for the birth of healthy children and for such medical supervision and environmental care as will keep them well from infancy through adolescence.

Such provisions as are necessary to carry out this program should be made available to every child in each community through the activities of the child's natural caretakers, its parents, or guardians, and the employment of a supervising physician, or, these failing, through the interest and support of the community itself.

It is at once recognized that few indeed are the communities in which this desirable goal is attained. Needless illness and death among children is a daily occurrence, especially among those born to parents less informed and of limited resources.

Our failure thus far to care for all our children satisfactorily should not be a cause of discouragement, for great progress has been made along the way, but it should rather be an incentive to further effort and should lead to frequent revision of the methods employed with the purpose of making them more effective.

The health of the child is promoted by any general measures which affect the health of the community. The introduction of a safe milk supply, the sanitary disposal of sewerage and waste, the control of communicable disease, the opening of parks and recreation centers in congested districts and improvements in housing conditions are all reflected in a lowered infant morbidity and mortality rate and in the betterment of the health of older children.

In addition to these general public health measures the dependent infant has other needs which are embraced in a complete child hygiene program. They may be chronologically enumerated as follows:

(1) Healthy parents, fathers and mothers free from damaged strains and untoward conditions which may injure their offspring.

(2) Adequate care of the mother before, during and after child-bearing.

(3) Medical examination and supervision (including proper diet and care) of
  (a) The infant
  (b) The preschool child
  (c) The school child; and of the
  (d) Child in industry

(4) Early treatment of incipient disease and prompt removal of remediable physical defects.

This program is the right of every defenseless child, irrespective of its race, color or the financial status of its parents. The well-to-do and informed parents can be expected to provide for themselves and their children the kind of attention suggested. It must be understood, however, that a large proportion of the illness and death among children occurs in those families where parents or guardians are indifferent to or unable to furnish the medical supervision and care desirable. In these circumstances does not the community have a responsibility in the matter and how can it be discharged? Naturally in matters of health, in the planning and setting up of measures necessary to promote personal hygiene, including the health of children, the community looks to its physicians for advice and leadership as it would appeal to its business men for help in the inauguration of a financial project.

The medical service intended further to reduce illness and death among all children cannot be carried on by health officers. Their number is far too small and only a small part of the country has an effective health organization. It is to the medical profession, present and future, that each community should turn for guidance. Are the 150,000 doctors in the land ready to lead in this tremendously important project? Thus far their training and practice have tended to make them individualists. They have been taught in the schools to study disease, its diagnosis and treatment as it is found in individual patients. The volume of good to suffering humanity that has been rendered by the general practitioner of medicine and surgery and by the various special branches is simply incalculable. The doctor, from the beginning, has given freely of his time and skill not only to those who could repay him, but also to the poor. This service usually has been rendered as a personal matter in response to a special appeal for assistance. It has been at best a haphazard method, however helpful to the patient receiving it. Rarely the medical needs of a whole community have been considered and steps taken by the organized profession to lead the

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community to adopt a program intended to meet these needs. In proof of these assertions one has only to refer to the fact that more than one-half of the maternal deaths occurring in child-bearing take place among women who have had no prenatal care;

That in more than 50 per cent of infant deaths occurring between one and twelve months of age, no doctor sees the little patient before the fatal illness;

That deaths from gastro-intestinal disease in infants under one year in the United States number about 15,000 annually;

That among rural children only 1 per cent under one year and 18 per cent under five years are vaccinated against smallpox, and among the same children but 3 per cent under one year and 29 per cent under five years are protected from diphtheria;

That thousands of our boys and girls are beginning their long educational careers handicapped by physical disabilities which should have been detected and removed years before. Certainly our profession has not done all it can to insure the physical welfare of the nation's children. It has not to the full accepted the responsibilities which rest upon it as the custodians of medical knowledge. I yield to no one in my admiration for the family doctor, but I would limit the term to that kindly physician who assumes the medical care of the family, who gives the mother the necessary attention before, during and after her confinement, who directs the feeding and regime of the baby through its infancy, protecting it at the proper time against smallpox and diphtheria, even though not called to do so, who examines from time to time the toddler to satisfy himself that the child is not choked with large tonsils and adenoids, that his diet is sufficient to insure good teeth and firm bones, who advises the whole family if a communicable disease arises and as far as possible prevents its spread and who has a sense of responsibility for the health and development of each member of the family.

Ought not the descriptive name, Family Doctor, to be reserved for physicians who render this kind of service and not be given to those who only answer "calls?" It is perhaps utopian to expect that every family in a community can be cared for by a real family doctor. It is for those whose health is not so supervised that the community, directed by an organized medical group, should provide. That proper professional care will diminish illness and death is abundantly proven.

Thus, in the private practice of a skilled pediatrician involving about three thousand patients, many of them infants, there were but 11 deaths, practically all of them from conditions that medical science has not learned to control. Similar results are being obtained among children attending regularly many of the infant welfare stations where medical advice is combined with nursing supervision in the homes.

John Dewey has said that "What the most intelligent parent desires for his own child, that must the community desire for all its children."

The best informed and progressive citizens in every community do desire to have all the children concerned properly born and healthily reared and they look to the local medical profession to indicate how this can be accomplished. Naturally the older busy practitioners, whose experience in the medical schools has been confined to the study and treatment of disease in the individual, are slower to acknowledge the idea of community responsibility for health. To this general statement there are thousands of notable exceptions. Many men of older years in active practice have seen the dawning of a new day and are giving of their time and energy to the support of various measures for the promotion of community health. It is to these socially minded doctors that much of the progress in public health, including child hygiene, can be traced.

The future of community health, however, is to be in the hands of the young men and women who will enter the medical schools hereafter. The part they will play in supporting communal health programs will depend largely upon the emphasis that is placed upon these activities during their professional studies. They should be informed at this time that every graduate in medicine, because of the knowledge acquired and the historic ideals of his profession, has a duty not only to care for the individual patients who consult him, but an obligation also to all the members of his community, and that he should be advised to join with his fellow physicians in support of approved measures intended to reduce disease and promote health, especially among the dependent groups. If the lay citizens, interested in promoting the health of their community, could receive cooperation and direction from all practicing physicians, approved public health measures could be under-

taken with more hope of success. It is with this in mind that in the opinion of the writer certain subjects and emphases should be given a larger part in the curricula of our medical schools.

It is greatly to be desired that our medical students, irrespective of the branch of the profession which they may subsequently follow, receive during their course some instruction in various forms of community sanitation and in the interpretation of vital statistics, the bookkeeping of humanity, not that they should become expert sanitarians, but that they be prepared to further intelligently these projects wherever their lot may be subsequently cast.

The student of medicine should have more instruction in the science of heredity, especially in its practical aspects, and should understand the possibility it presents of improving the quality of our racial stock. He should be told that one of the most important questions having to do with child hygiene in its wider aspects is "Who are to be the parents of the next generation?" He should know that the infant mortality rate, other things being equal, is lower among children of long-lived parents. He should be informed of the great loss the nation sustains in its disregard of negative eugenics, from its indifference to mating among the notoriously unfit. He should be prepared as a physician to stimulate in the community a desire to improve the racial strength of the next generation; he should be taught to study his families as well as his patients. He should be made to realize that at present much of the labor of the pediatrician is thwarted because it is expended upon children with defective inherited strains, the result of the neglect of the established laws of heredity.

Again it should be emphasized in his course in obstetrics, when he is taught the diagnosis of the various complications of pregnancy, the importance of a complete physical examination, of pelvic measurements, of blood pressure determination, of aseptic technic and the other matters that are included in essential prenatal, natal and postnatal care, that this kind of service is not available at present to many women except at a price they cannot afford. He should be encouraged to take not only meticulous care of his own patients, but to strive also to devise a practical plan by which good, clean obstetrics may be brought within the reach of all women in the area of his practice. He should be reminded that the measures which result in better care of the mother are most effective also in reducing the number of stillbirths and deaths among infants in their first month.

And again, instruction in pediatrics in recent years is assuming that place in the curricula of our best medical schools that it has long deserved. The anatomy and physiology of the infant, the factors concerned in its growth and development, its nutrition, its environmental needs and the various diseases, including the communicable diseases, prevalent at an early age period are considered, but largely from the standpoint of the individual patient.

There is room in many medical schools for increased emphasis upon methods of bringing every child within the radius of the doctor's influence, that it may obtain the supervision and protection which are essential to its welfare.

Similarly in the runabout child between the period of infancy and school, its rapid physical and mental development, its susceptibility to infection, its frequently occurring physical defects, all suggest the importance of stressing to the student that when he begins his practice he should be active in attempting to bring all preschool children under medical oversight.

In maternal and child hygiene the knowledge of how mothers and children can be saved has so far outstripped medical community practice that various procedures to meet the situation have been begun often by lay organizations with the assistance of certain members of the medical profession. Much pressure is made upon health officials to enter this preventive field thus far not occupied by organized medicine.

A practical method of bringing professional assistance to those mothers and young children who would otherwise be without it are the so-called maternal or prenatal and the child health conferences supplemented by visits in the homes by public health nurses. Through these means thousands of mothers and children are being reached. At such conferences medical histories are taken, physical examinations made and preventive advice is given. Mothers who attend the prenatal conferences are referred to a private physician or a hospital for delivery. Children in need of treatment who are examined at the child health conferences are often sent with a note to a doctor or hospital.

These conferences, the operation of which has become fairly well standardized, should be made the subject of special study in the obstetric and pediatric departments of our medical colleges and, when possible, students should be required

to work in them so as to become familiar with the methods employed and with the relationship of these services to the private practitioners and to the needs of the community. Both the prenatal and the child health conference, if this is the accepted local plan, should be carried on by the organized profession of the community, or at least with their knowledge, approval and assistance. Other plans may be better adopted in various areas, but the end in view is the same, namely: that adequate obstetrical care may be made available to every mother in need and that every child not receiving it shall have the opportunity of medical supervision.

We are all aware that this goal is not everywhere attainable. Considerable numbers of our people live in widely scattered homes with difficult problems of transportation and out of reach of medical assistance. Some years ago the Appalachian Mountain region would have been listed as such a territory. Here no adequate professional service seemed possible for the isolated settlers in their lonely cabins until the Frontier Nursing Association began its work. This and similar humanitarian endeavors for bringing to the less fortunate the blessings of the preventive and healing service should be made the subject of sympathetic study in every medical center.

The methods of supervising the health of children of school age are perhaps more clearly recognized, but are rarely found in completely satisfactory operation. They include the sanitation of the school premises, the examination of and advice to the teacher and pupils and the health instruction of the students. This service is invaluable not only to the educational authorities and to the public because of the danger from infectious diseases through school children, but to the health officials and practicing physicians as well. Many of our schools are at present without adequate health control. It could be greatly extended through the active cooperation of the medical profession.

The value of school hygiene should be stressed in the medical school. Similarly the examination of the adolescent children engaged in industry and the sanitation of the shop or factory in which they work should be a part of the information possessed by physicians when they begin their practice.

We have set up and attempted to support the thesis that the ideals of child hygiene will not be approached until every community under the leadership of its adequately trained physicians sets up a working program which makes available to all dependent women and children preventive advice and medical supervision.

It is realized that even when this is done (how far we are from this first step) only a real beginning will be made. There remains the long, slow education of the public, the persuasion of the indifferent husband that his wife needs prenatal care, the bringing home to ignorant parents the value of periodic examination of their growing children, the importance of proper food, abundant sleep and habits of cleanliness, and so through the whole gamut of admonitions which become so trite to the intelligent before they make any impression upon the careless. It is in this educational field that the services of the health official and nurses are most effective. Part of their job is to advertise the services that the organized profession should make available.

The point to be stressed, however, is that until there is some organized plan to furnish preventive supervision to those who cannot provide it for themselves, much of the educational propaganda of health officers, nurses and other agencies is visionary and academic. How can the underprivileged family learn of the advantages of personal hygiene and medical supervision unless they are told, and if told, how can they profit by their instruction unless there is a recognized community plan to bring these services within the reach of all who cannot provide them for themselves? The abuse of free advice by those who can pay can be prevented by a local social agency or otherwise. On the other hand, it is a frequent experience that families of moderate means learning of the value of community health services prefer to secure similar care from their own doctors.

Such a service for the community, under the leadership of the medical profession, should contemplate a fair remuneration to the doctors who are actively engaged. This could be arranged by agreement between county commissioners and the county medical society. Some assistance might be expected from state departments of health. The plan might include payment to outside physicians who were called in to assist as consultants. It is needless to emphasize that wherever health clinics have been properly conducted an increased number of patients consult physicians, and often for conditions they can remedy, than would be the case without the clinics. Moreover, if a community begins to think in terms of health preservation as well as

in terms of the treatment of existing disease, those physicians who are equipped to render preventive service will more than make up the income lost from the reduction in typhoid fever, diphtheria, and infant diarrhea.

In this plea for a medically directed health service it is not expected that all doctors of a community should give much of their time to the enterprise. It is necessary, however, that they approve it, support it in their meetings and before the public. In my own State outstanding contributions to public health projects, one resulting in greatly improved provision for our insane and another in raising the nutritional standards in our almshouses, have been brought about through the initiative of two specialists, urologists, who continued meanwhile their active practices.

One must be blind who cannot see that a new era in medical practice is under way in this country, one that emphasizes prevention as well as diagnosis and cure, one that encourages research but demands the general application to all citizens of the proven results of investigation.

Certainly much assistance is being given to the movement by physicians at present in practice. Community health, particularly child health, has been stressed through postgraduate courses and seminars held in medical centers and has been proclaimed by carefully chosen teachers who have met the doctors in their home territories. Infant welfare is a frequent topic of discussion at local medical meetings.

All this has raised the level of the stream, but tide of adequate care for every child throughout the nation will not become flood until all the members of our great profession emerge from the medical colleges socially minded, prepared to do what they can to bring the benefits of preventive medicine within the reach of all. How health officers would welcome this support! On this account one turns to the various medical schools of the country with hope and expectation.

In going over the catalogues of our leading medical colleges one finds with satisfaction that a majority of them require one or more courses in public health and some, a sanitary survey with thesis of a town or industry before graduation. In a few, students are expected to work in child welfare stations, and in fewer still, through actual visiting, under supervision, they are enabled to become familiar with the sanitary conditions in rural homes. All this is excellent and indicates an increasing stress upon community health. In some of our better schools, however, students may complete the course, graduate and enter the practice of medicine with little or no knowledge of the recent developments in public health and without appreciation of their duty actively to promote it.

Although the value of adequate care of the mother and the preventive care of infants and young children is taught in the obstetrical and pediatric departments, judging from the official information available in only a few of our medical colleges of high standing, is the obligation of organized medicine to foster community child health in a practical manner given formal recognition. If this obligation were understood and accepted by every graduate in medicine, an effective child hygiene program could be established in every area in the country and the present unnecessary illness and death among children could be greatly reduced.

DISCUSSION (*Abstract*)

Dr. L. T. Royster, Charlottesville, Va.—Dr. Knox has brought to our attention and stressed facts which every physician should not only be familiar with, but should be prepared to apply in his daily practice. I wish to correct an impression which prevails rather generally and state that pediatrics is not a specialty. It is an attitude of mind, a method of procedure; it has for its ultimate end the attaining of the well being of the adult that we visualize in the child under our care rather than the treatment of the actual present acute condition or disease alone. Viewed thus, every physician who attends children at all, in whatever capacity, should practice pediatrics.

Preventive medicine can be applied most satisfactorily during childhood; the adult can in most instances only be patched up. Therefore, in a broad sense preventive pediatrics and preventive medicine are identical. It has been said that prevention should begin at the time of the birth of the child. I think we should rather say that preventive medicine for the infant should begin at the moment of the infant's mother's birth so that at least when a female infant is born into the world we should begin to consider preventive measures for her offspring a generation hence so far as obstetric trauma is concerned. What bearing, therefore, has these considerations on the teaching of pediatrics in medical schools. We all know that it is hard to teach old dogs new tricks, and it is difficult for the man who has been out of medical school ten or fifteen years to readjust himself to new thought. Accordingly, it is evident that if we wish the profession to be child-minded an impression must be made on the undergraduate medical student. We teachers of pediatrics certainly should teach every medical student what the rights of the child are and what his obligation is to the child regardless of what specialty he may practice.

The White House Conference stressed an old principle in novel language, using the expression "the appraisal of the child." Appraisal of the child cannot be made in a single examination. To be sure, a physician of experience can in most instances determine whether that child is free from acute and latent disease, but he can hardly determine what is normal in a single examination; normality must be measured in terms of progress, in terms of the process of maturation. Repeated examinations with comparison of the findings made at each individual examination are, therefore, necessary. A fault of many medical schools is that students are not adequately taught what constitutes a normal child and normal growth. Most physicians know relatively little about how a child should progress, at what rate it should grow, at what rate it should put on weight, at what rate the bones and the muscles and the viscera normally grow. Nor are they familiar with the normal emotions. Medical students should be taught normal psychology, preferably in the course on pediatrics. The physician should familiarize himself with the approximate time of the appearance of emotions so that he will be prepared to advise mothers when the child develops a new slant or bias. Many children are called problem children who show a mere exaggeration of normal emotions. Unless the physician knows the normal he cannot know the abnormal.

We do not expect to accomplish a great deal in a very short time. I well remember when an operation for appendicitis was looked upon as a last resort. Likewise in diphtheria the administration of antitoxin was a last resort. Today we know that as soon as the diagnosis of appendicitis is made an operation is indicated, and when the diagnosis or even the suspicion of a diphtheritic process is in our mind, the administration of antitoxin is the only proper procedure. Such a viewpoint covers a generation of development. The question of the prevention of disease in children, not only through the efforts of the individual physician, but through the efforts of groups of physicians, is now a social one; the physician must be socially minded as well as child minded.

The stability of any structure is dependent on the strength of the foundation. The structure of society is no exception. Therefore, the foundation must be made strong through the children upon whom the superstructure of society must rest. Make and keep the children well physically, mentally and morally and the social order will be maintained in a state of more nearly stable equilibrium than it is today.

Dr. Harvey F. Garrison, Jackson, Miss.—Of course, we do not hope nor do we expect to make pediatricians out of all students or practitioners of medicine, but we do hope to see the day where there will be considerable improvement in the training of physicians in the promotion of child health.

I hope that every pediatrician present at this meeting will endeavor to discuss this same subject before every medical society in his state in the near future.

Dr. Knox (closing).—An ideal program in child health for every community will be attained only in the far future, but those of us who are interested today in promoting the health of the child must understand the difficulties and take steps to remove them. Unquestionably in setting up a program such as has been advocated, the practicing physicians must play a leading role. If they graduate from their respective schools of medicine with a sense of their responsibilities as doctors in the support of preventive measures, whether or not they become pediatricians or health officers, a long step forward will be taken toward a goal. The present generation of doctors, graduates of the medical schools of the country, all have influence as alumni in their respective schools and can do much to secure the teaching of prevention, including child hygiene, as an essential part of the curricula.

AUTISTIC THINKING*

By W. R. HOUSTON, M.D.,
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Autism is a term applied to the thinking of the schizophrenic. The schism in the thinking of the schizophrenic is a cleavage between wish and reality. Regardless of the admonition of reality, which presses in upon him with relentless insistence, the schizophrenic believes that his wish has come true. Taking no thought of reality, renouncing all heed to the lessons of experience or the teachings of logical criticism, he thinks as we think in dreams. In his magalomania he sees his wishes fulfilled, in his delusions of persecution he projects his own incapacity upon his environment. It is not truth that he seeks, but the fulfillment of his wishes.

It is a principal glory of modern psychology that it has ceased to emphasize the old scholastic terms, "intellect," "volition," "conception," "representation," and has centered attention on the wish as the dominant issue. In the great vital tendencies, in the fundamental biological trends, the wish takes its root; it finds it flower of fulfillment in belief. This truth finds its expression in many a popular phrase: "We believe what we want to believe," "Men's opinions vary with their interests," "The wish is father to the thought." Are we to gather from these phrases that mankind has always recognized his beliefs and thoughts to be merely wish fulfillments?

As long as 2,500 years ago, Confucius recognized that thinking was of two kinds, which he designated agglutinative thinking and root-and-branch thinking. In current phraseology we call these two types of thought autistic thinking and thinking in terms of reality. The practical thinking of the world is in terms of reality. The carpenter building his house, the merchant dis-

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tributing his wares, the engineer planning his bridge, the physicist setting up his experiment, the chemist, the mathematician, the astronomer, if they are to think to any purpose, are thinking in terms of reality. With the advance in man's knowledge of his universe, the territory in which autistic thinking can disport itself has been encroached upon and grown progressively less. When pestilence rages in the camp, we no longer burn hecatombs of oxen to avert the wrath of an angry god; when a shadow begins to cover the face of the sun, we no longer beat gongs to frighten away the angry dogs of heaven; few still hope to bring rain or avert the hurricane by incantation and prayer. With each decade we see some ground gained for realistic thinking, some diminution in the scope of wishful thinking.

Yet none will question that there remains a great body of belief and thought that is both active and influential, yet that is wholly autistic, dreamlike, wishful. Can this autistic thinking that is observed in persons who go about their business daily and occupy places of importance in the community be distinguished in kind from the autistic thinking of the schizophrenic? By what criteria can we separate the delusion of the insane who is under custodial care from the delusion of the citizen whom we call normal?

It is argued that a delusion is insane when it is not accessible to correction by logical reason based upon reality. Such a distinction is plainly invalid. Attempt to prove to a devout follower of Mrs. Eddy the existence of disease by facts adduced from pathology and parasitology. Attempt to prove to a devout Jesuit that bread and wine, whatever words were said over it, remains bread and wine, and one becomes at once aware that the tenacity with which a delusion is held does not make it an insane delusion. The believer in ghosts and spiritualistic visitations is inaccessible to realistic logic and may yet be a useful and even brilliant member of society. Yet his belief has as little support from reality as the belief of the schizophrenic who fancies himself the Messiah.

Since we must seek some other grounds than the tenacity with which a delusion is held to distinguish the delusion of the sane from the delusion of the insane, it is suggested that the delusions of the sane are harmless, while the delusions of the insane bring them into conflict with ordered society. A very brief examination of history will show this distinction to be without foundation. Consider the cruel deprivations and penalties inflicted by savages out of respect to their taboos; consider the populations wiped out, the regions laid waste through the stubborn adherence to their idol worship of those who refused to accept the teachings of Islam; consider the bloody martyrdoms, massacres, and wars that Christian people have endured for some slight difference of opinion as to the constitution of the Trinity. From these instances we must conclude that a belief which owes all to the emotionalized wish that begot it and nothing to reality may be very destructive to the welfare of the believer and yet cannot be reckoned an insane delusion.

If it is not the tenacity with which it is held, and not the harmfulness of the delusion that define it as insane, what is it that makes us reckon the delusion of one man compatible with sanity, and of another an indication of unsound mind? Evidently the only valid point of differentiation between the delusions of the sane and the delusions of the insane lies in the singularity of the insane delusion. A delusion that is held by one man and not shared by those about him we consider an insane delusion. Mankind has always been severe with singularity. Most monstrous delusions, that have led to the immolation of children to Moloch, to the human sacrifices of the Aztecs, to the burning of witches, were held in their day to be excellent orthodoxy; but the innovator, the discoverer, the seer, by reason of his singularity, has generally fared ill.

We must agree, then, that there is no difference in kind between the autistic thinking of the schizophrenic and the autistic thinking of the average citizen. Shall we say, then, that delusions are in themselves bad and to be condemned? Would we do away with all autistic thinking and think only in terms of reality? Or may it not be that there are some good delusions that should be kept and fostered? Is it not true that we must judge each delusion on its own merits and decide whether it is a delusion that should be combated? The answer unquestionably is that autistic thinking, as such, cannot be condemned. There are delusions which make for happiness, for better and fuller living. Without the delusions of hope we could scarcely support life. We feel kindly toward the delusions of youth. Our warmest loyalties are sustained by delusions. The delusions of love, of motherhood, of patriotism, not only make the poetry and romance of life, but contribute to its se-

curity and preservation. A man who lived without delusions would be a cold monster.

These are reflections prompted by a reading of Bleuler's charming and spirited book entitled "Undisciplined Autistic Thinking and Its Correction." In this book Bleuler sharply arraigns the medical profession on the charge of autistic thinking. It would be impossible to take issue with Bleuler on the point that a very large proportion of medical thinking, particularly in the field of therapeutics, is undisciplined and autistic. He shows by countless pregnant examples, examples which may be readily paralleled by any acute observer, that a large proportion of all therapeutic thinking is imprinted with the same marks of autism that we commonly criticize in our schizophrenic patients. We find it heavily charged with the same naiveté and credulity that were found in the childhood of the race.

I find myself, however, compelled to differ with this eminent authority in the assumption he makes that it is sufficient to say, in condemnation of a belief, that it is autistic in character and that it reveals a type of thinking which is found in the insane. Autistic thinking is found among the schizophrenic, to be sure, but it is also equally characteristic of every day human thinking. The man who thinks with clearest grasp of reality in one field, in another indulges in an orgy of undisciplined autistic thinking. Eminent physicists like Lodge and Crookes, though rigorously logical in the serious undertakings of the laboratory, become at once as credulous as Hottentots when entering upon the investigations of spiritualistic séances. They abruptly pass from controlled thinking in terms of reality to autistic, wishful thinking. When we see them go with such facility from root-to-branch thinking over to the sticky, agglutinative sort, we recognize that these two types of thinking go on quite commonly side by side in the same head. This is a great misfortune because the inference is readily drawn that, since a man has thought clearly in terms of reality in one field, his thinking in another field must carry with it the same characteristics of sound logic. It must be confessed that, as the human mind is constituted, its several operations may be carried on in logic-tight compartments, realistic thinking going on on one side of the partition, autistic thinking on the other. Perhaps the very impermeability of these compartments is evidence of sanity, since we are accustomed to think that the insane delusion comprises the entirety of the cortical reaction. Lodge and Crookes carried no part of their mysticism into the world of physical investigation, but it is not so sure that a paranoid schizophrenic would not allow his insane delusion to disturb his thinking as carpenter or husbandman.

Autistic thinking is a part of normal thinking; it is so much a part of normal thinking that it has been incorporated into the education of children. Children are taught delusions in the form of fairy stories, *Märchen,* Santa Claus legends and the like as forming a desirable part of their mental hygiene. Certain grave educators, impressed by the need of the race to think in terms of reality, have objected to the fact that children are told these stories. In my judgment, it is an excellent thing that they should be instructed in delusions in order that, if possible, they may early learn the distinction between autistic thinking and thinking in terms of reality. It is very desirable that early in life they distinguish reality from delusion, that they understand the process by which one awakens from delusion, that they become acquainted with the painful shock of seeing a delusion shattered, that they become inured to an experience that they will have to rehearse many times in life; by tasting early the bitterness of knowledge and the pain of being shorn of a delusion they will learn to thrust ruggedly on, learn to esteem delusion as a childish thing to be put away, learn to face cold, unemotional reality as the fit attitude of maturity.

I have attempted to show that delusions are to be adjudged bad, not because they are autisms, but because of their effects. What delusions may prevail in the fields of economics, of politics, of metaphysics, it is not our concern as medical men to discuss. It is our business to concern ourselves closely with the autistic thinking that prevails in the field of therapeutics. Much has been accomplished in the direction of banishing autistic thinking from the domains of physiology, biochemistry, pathology, etiology, diagnosis and all the fundamental approaches to medical practice. It is when we come to the final step, the application of the medical sciences to the uses of the sick man, that we are prone to make the sudden jump into autistic thinking. Therapeutics is the chief business of every physician. Yet while it is a truism that treatment is the physician's chief concern, how much time, how much attention, is given to this subject in our medical schools? Therapeutics

has to a large degree been abandoned to autistic thinking. Is there a medical school in the country that has a department of therapeutics, with a professor of therapeutics at the head of it? Is there an institution or a foundation devoted to therapeutic research? Is it not true that the professor of medicine, after having devoted fifty-five minutes of his clinical hour to problems of etiology, pathology and diagnosis, may in a cursory and half-apologetic tone say something in the last five minutes concerning treatment? Is not this the explanation for the generally observed fact that while problems of diagnosis are dealt with as matters for clear, logical thinking, kept in close contact with experience and reality, at the same time treatment, the greatest problem of all, remains in a logic-tight compartment, at the mercy of autistic thinking? It is just because it is well understood that it is a subject abandoned to autistic thinking that the leaders of our profession rarely concern themselves with treatment. Look over the table of contents of the *Archives of Internal Medicine*, or the *American Journal of Medical Sciences*, and observe how many studies of treatment are to be found; perhaps four or five in a hundred contributions, and these few, as likely as not, marred by lapses in logic and obvious uncroppings of autistic thinking.

Our pharmacopeia has been to a great extent the repository of the autistic thinking of previous generations. Half the drugs listed in it came down to us from the Arabic physicians. In addition to these books of curious remedies, the inheritance of autistic thinkers of the past, chemical factories are pouring out an increasing stream of new preparations, mostly nerve poisons of one sort or another, and by alluring advertisements and suave salesmen we are invited to a feast of autistic thinking concerning these. As you well know, much of our therapy is psychotherapy, but it is not clean-cut, clear-eyed psychotherapy conscious of its aim and in search of the shortest path to attain that aim. It is psychotherapy masked and camouflaged by a smoke screen of drugs and prescriptions.

It is true that we are making progress. Something has been done on the Augean task of cleansing the pharmacopeia. No one pays much attention to the National Formulary, despite the great names signed to some of the complex prescriptions embalmed there. Our Council on Pharmacy and Chemistry has done a great work and has placed us in a better position to escape autistic thinking than is enjoyed by the countries which have no such Board. The forthcoming revision of the pharmacopeia is to delete a hundred preparations from its pages. All this is worth much; yet with all that has been done, we are still far from thinking in terms of reality. Whether through autism or through ignorance, we are largely unaware of the distinction between psychotherapy and drug therapy. Think of the vast sums spent on ovarian tablets, corpus luteum pills, which recent studies have shown to be utterly inert. Think of the seas of mouthwashes prescribed, Dobell's solution, liquor antisepticus, glycothymoline, boroglycerine, and what not, the psychic antiseptics, as Sollman calls them. Think of the enormous tribute paid to advertising skill, the common willingness to pay five or ten times as much for a proprietary name as for the same value by the name offered by the U.S.P.

I cannot extend this story, but I have sought this opportunity to remind the gentlemen of this Section, accustomed as you are to deal with psychic problems, that your brethren of the profession are in need of your care. I appeal to you to leave your psychic derelicts and give thought to the care of those in your own household, as your eminent colleague Bleuler has so manfully tried to do. While no important advance of the race has come through autistic thinking, I am prepared to concede that such thinking has its place. Its place is not, however, in the field of therapeutics. There autistic thinking is noxious and destructive.

But your task, whether you approach it as psychoanalysts, as moralizers, or as persuasive educators, will not be easy. You attack autisms highly charged with passionate emotion, as is all autistic thinking. Your weapon is disinterested thinking in terms of reality. You oppose to the powerful reactions of the viscera the feeble control of the cortex. Yet the cortex has won many conflicts against visceral thinking. Autistic thinking, even in its favorite stronghold of therapeutics, may finally meet defeat.

DISCUSSION (*Abstract*)

Dr. Hugh J. Morgan, Nashville, Tenn.—I have given a good deal of thought to the teaching of therapeutics. I am afraid that I am guilty, in teaching, of devoting "fifty-five minutes of the clinical hour to problems of etiology, pathology, and diagnosis" to the neglect of treatment, and in most instances I do this deliberately. The teacher of internal medicine has such a broad field to cover that it would seem to me to be unfair to expect of him to try to impart to his student much

of the technic of psychotherapeutic practice. Courses in psychology and psychiatry should give the intelligent student the foundation for that development of understanding and insight into human nature which makes for successful psychotherapy. But this must be complemented by experience with patients. The earnest and intellectually honest practitioner uses psychotherapy constantly and to the enormous advantage of his patient and himself. The indifferent or stupid practitioner continues his autistic therapeutics and I doubt that there is anything that we can do about it. What Dr. Houston really is asking for is the inclusion in the curriculum of more studies dealing with the human mind and with this request I am in full accord. Nevertheless, I am inclined to think that he is using the present-day medical student and younger practitioner as an innocent, sacrificial victim in order to send an inconspicuous arrow in the direction of some of the older and perhaps less sophisticated transgressors.

Dr. W. W. Young, Atlanta, Ga.—All of us at intervals need something arresting to make us attempt intelligent thinking. We are too prone to rationalize. The doctor with his tendency to feel the urge that he must "do something" falls the victim of this type of thinking most frequently. Too much therapy is cast in this frame and too little intelligent reasoning is used. The individual as a distinct problem should be the starting point of reasoning.

ADVISABILITY OF REPAIRING OLD LACERATIONS OF CERVIX AND PERINEUM AT TIME OF SUBSEQUENT DELIVERY*

By JAMES R. BLOSS, M.D.,
Huntington, West Virginia

The end-results of obstetric service are as yet far from perfect. Probably the obstetric millennium will never be attained. That conditions can be improved greatly is self-evident. The economic condition of the patients, an ignorance of the highest type of service on the part of the laity, and a complacent acceptance of least exertion as the easiest course on the part of the physician, all play a role in the situation which confronts this section of our profession. The opinions and conclusions expressed in this paper are the result of the evolution of the author's personal obstetric experience.

It is agreed that much of the invalidism and semi-invalidism of women and the etiology of more than half of the gynecologic surgery dates from childbirth. This does not consider the economic loss to the family nor an appreciation of its influence upon the marital happiness of husband and wife, and thus its status as an etiological factor in divorce. Anything which will be of help in correcting unsatisfactory natal or postnatal results should be welcomed with open arms and unhesitatingly adopted.

More than thirty years ago, during my student days, we were taught that it was not advisable to repair lacerations of the soft tissues of the birth canal immediately after delivery. We were advised to wait until the third to seventh day. In my early years of practice this advice was followed. Results were very disappointing and discouraging.

Since I knew of the operative work of the late Joseph Price, of Philadelphia, in the homes of patients, it seemed to me that it should be possible to secure better results by immediately repairing the injuries sustained during delivery. This plan was adopted and the results showed improvement, but still did not satisfy. It was found that a few sutures placed through the tissues *en masse* did not so approximate the deeper structures that union could be reasonably expected. It was but a step to repairing in layers, burying the sutures of the deeper planes separately. The result with this type of repair was much more gratifying.

At the beginning of this method of repair it was noted that where there was a relaxation of the perineum due to laceration from previous deliveries it was possible, at a subsequent birth, to repair it with a fair assurance of a satisfactory outcome. This procedure was only carried out, however, if there was a new laceration.

From this it was but another step to undertake the repair even though there was no fresh tear of the mucous membrane or skin. Results were more satisfactory. There was as yet no attempt made to repair old lacerations of the cervix. This reparative work was done in the homes of the patients as well as in the hospitals.

In August, 1925, there appeared an article in the *American Journal of Obstetrics and Gynecology* by J. L. Bubis, of Cleveland, on this subject. The reports given by him were based upon not only old perineal lacerations, but also those of the cervix, herniations of the vaginal walls (cystoceles, rectoceles and so on) and hemorrhoidectomy as well.

Considering his report in the light of my own experience, it seemed that his conclusions were sound and his recommendations justified. Since that time repair of the cervix, rectocele and the perineum has become practically a routine. The

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end-results are not perfect in all cases. It would be too much to expect that. Seventy-five per cent, however, get relief from leukorrhea, backache and so on, that is, from the symptoms due to loss of support by the pelvic floor and the infection of old cervical lacerations. Twenty per cent show improvement, but still have symptoms due to displacements, either retroversions or prolapses. These can be relieved only by intra-abdominal procedures for their correction. Five per cent possibly will fail to secure union of the repair of the levators and the perineum. In most of these last the poor result is, in my opinion, due to too early resumption of their household duties, a postoperative neglect in seeing that the fecal contents are not allowed to accumulate in the rectum, finally, and certainly not the least among the causes of failure, too early resumption of sexual relations.

Certain objections have been offered to this puerperal gynecology, as Bubis terms the plastic repair work. It is said that the tissues are so swollen and contused that it is dangerous to try to suture them. Then comes the objection that the sutures will not hold because of the friability of the tissues; that they will cut out. The next objection is usually that because of the contusion and laceration it is not possible to dissect out the various structures with any degree of anatomic accuracy. It is also advanced that the danger of infection is too great to justify the undertaking of this plastic procedure. Finally is the suggestion that there will be almost always sloughing to destroy the efficiency of our surgical endeavor.

Personal clinical experience leads me to conclude that none of these objections is tenable. Grouping the first three and beginning with the assertion that it is not possible to identify the various planes of tissue because of edema, contusion and so on, it can only be replied that if one knows the anatomy of this area; knows what he is looking for and where it normally should be, and then is gentle in handling the tissue, it will be quite a revelation to find how readily the structures are identified. That there is a danger of perforating the rectum or bladder with a needle while repairing a rectocele or cystocele after delivery with contused and softened tissue must be admitted. I have seen these accidents happen to exceptionally able men when doing this repair work electively and not after a delivery. My contention is that the danger is no greater after delivery than at any other time. Experience has not convinced me that these tissues will not bear suturing because of friability. The secret of this, it seems to me, is gentle and snug approximation only, with no undue tension on the tissue by tightly tied sutures.

That there is danger of infection cannot be denied. This danger, however, is the same one which is always present in all surgical undertakings about the vagina, namely: the introduction of additional strains of bacteria. It must be insisted upon, of course, that in the presence of an acute vaginitis due to bacterial invasion by the gonococcus, or any other pathological type, no repair work be undertaken. Such a procedure would be foolhardy. We do not hesitate to remove with scissors or a knife the margins of lacerations of the cervix even if they are cystic and chronically infected. Occasionally the whole margin of the cervix should be trimmed off for this reason. The drainage provided and the fact that the patient has developed an immunity to her own particular vaginal bacterial flora, accounts, in my opinion, for the rapid and uninfected convalescence of these patients. It must be insisted that the technic of the operator's personal and operative asepsis be above suspicion. Carry in no strange bacteria!

As to sloughing of the tissues, my experience is limited to two cases. In both there was sloughing of a small flap of skin just below the border of the vaginal outlet. Each might have been avoided had not a strenuous effort been made to achieve an artistically perfect cosmetic result. Both of these sloughs were entirely superficial, healed promptly, and in no way affected the reparative process beneath the superficial fascia.

The criticism that the lochial discharge will be irritating and prevent union, my experience shows to be unfounded. It must be emphasized, however, that great care should be exercised to approximate the edges of all intravaginal wounds to prevent undermining by the lochia. Unless this is done there is danger that pockets of discharge will interfere with union. Personally, it has not been found difficult to prevent this.

It is not advisable to undertake to make these repair operations if the patient has been severely shocked by prolonged labor and there is a tendency to uterine inertia. By the same token, if there has been a hemorrhage postpartum, or even an excessive blood loss without a definite hemorrhage, an extensive repair should not be undertaken immediately. In these patients, shock and hemorrhage should be combated as in any

other injury. Then we should undertake the desired surgical steps. At times it will not be safe for some hours or even days. These cases are the exception, however, and must be dealt with as the occasion arises, and the operator's experience and judgment may guide him.

This work has much to commend it. To begin with, there is the economical consideration. Any surgical procedure which can be safely and satisfactorily done during an enforced rest in bed, such as the convalescence after delivery, is surely a financial economy. In addition, we must not overlook the time economy as well, for these women are spared the several weeks of necessary convalescence from a later operation. A matter for consideration also is the fear of operation. Many women will drag along for years in pain and discomfort because they are afraid to undergo an operation. After a delivery with subsequent repair the improvement in their physical well-being is such that they are chagrined that they were not made to have the operation sooner. Since they did not have it earlier, and after it they feel much better than they have for years, they become ardent advocates of repair at the time of a subsequent delivery.

It is found that the process of involution is more rapid and more complete, if this term may be applied, after repair of old cervical lacerations. The profuse leukorrheal discharge from infected cervices is much improvd, in many instances cured entirely.

In those patients with rectoceles and relaxed or destroyed perinei the benefit in comfort and improvement of general health, mental as well as physical, is remarkable. The added physical comfort during, and the increased psychic satisfaction of sexual relations, is a point to be given thoughtful consideration.

Personal experience only leads me to summarize:

First, these operative procedures are feasible and safe.

Second, the end-results justify the added surgical manipulation under anesthesia necessary to carry them out.

DISCUSSION (*Abstract*)

Dr. W. B. Anderson, Nashville, Tenn.—As an obstetrician my main object and desire has been to deliver women and dismiss them in as good condition physiologically and anatomically as I received them. Dr. Bloss goes a step further, and according to his statistics, leaves his patient in a better condition than when he received her. Now the question is, is this the proper time to do corrective surgery? We all know, "In the interval of pregnancy a perineorrhaphy is frequently a bloody operation," and the same may be said of a trachelorrhaphy or of correcting a cystocele or rectoce'e. These parts are more vascular at this time than in the interval after pregnancy. I have seen more blood lost in repairing a lacerated perineum when I undertook to denude more of the surface than would be lost in an ordinary delivery.

However, there are times when this is justified. When there is a fresh tear in a perineum that is relaxed from a previous laceration, and this is rare, one is justified in straightening the jagged lines of his fresh tear and adding to it if it can be done without too much loss of blood. In doing this one should be prepared to suture the wound as he works, to prevent loss of blood. Now this is as far as I have felt justified in repairing old damage to the soft parts. Of course all primary damage to the perineum or cervix is repaired at once.

We all know that three of the main factors in inviting infection are lowered resistance, constitutionally and locally, and unnecessary manipulation of the parts which have open wounds. Loss of blood weakens constitutional resistance, besides rendering the mother's condition poor for nursing. The soft parts are already bruised and stretched, which weakens local resistance. There is no such thing as knowing that one is working in a sterile field.

As to repair of the old lacerated cervix: to begin with, I doubt that a backache is ever caused by a lacerated cervix *per se*. If the old laceration of the cervix is bad enough to cause a leukorrhea the appropriate time to correct it is after involution when a cervix looks like a cervix. The cervices which I have examined immediately after delivery have been so convoluted that I think I do well to pick out the primary tears. But why itemize these objections? Dr. Bloss' paper minimizes them and shows wonderful statistics to prove his ground. The most to be gained by such plastic surgery is the economy of a few days in the hospital, which I admit is an important item. But personally I think the ordeal of labor is enough for any woman at one time and I believe her prospect of nursing her baby will be better if this plastic surgery is postponed till after involution or until some time during the interval after pregnancy.

Dr. H. J. Stander, New York, N. Y.—It is not our practice to perform cystocele and rectocele operations at the time of delivery. We believe that such extensive operative procedures should be postponed until the completion of the puerperium, and we usually advise that they be performed nine months or a year following the delivery. The repair of a simple old laceration of the perineum is a different matter; here one may use his judgment as to whether such a repair should be done at the time of delivery or not.

Dr. Thomas Benton Sellers, New Orleans, La.—For the past few years it has been our routine practice to repair old lacerations of the perineum at the time of delivery unless there is some contraindication in the patient's condition or some complication at delivery. My end-results have been excellent and, I believe, superior to those obtained in elective operations.

I have done a large number of perineorrhaphies, but only a limited number of trachelorrhaphies and anterior colporrhaphies at the time of delivery. In spite

of the fact that in these cases there were no complications, it is my opinion that repair of the perineum, amputation of the cervix and anterior colporrhaphy all performed at the same time constitute too extensive an operation to be done at the time of delivery, although I believe that in selected cases perineorrhaphy and trachelorrhaphy can be safely done.

Dr. Bloss suggests amputation of the cervix, but I question the advisability of doing this just after delivery. In our practice conization of the endocervix by means of the electrosurgical unit has practically eliminated necessity for this operative procedure at any time.

I agree with Dr. Bloss that all of this repair work should be done only in the hospital by a well-trained, well-equipped obstetrician or gynecologist, as great damage can be done by the inexperienced. It is because of this danger, probably, that many teachers oppose the procedure of immediate repair of old lacerations.

Dr. W. T. Pride, Memphis, Tenn.—For many years I have repaired every perineum and cervix that needed it at the time of delivery. Several cases with rectovaginal fistula were successfully operated upon and cervices were amputated or repaired as the case demanded. I usually get better results at the time of delivery than at an interim operation. After all it is a matter of training and experience.

Dr. C. J. Andrews, Norfolk, Va.—There seems to be some difference of opinion on this subject. Probably the different speakers are thinking of different things. My conception of the matter is that rectocele and cystocele, often associated with prolapse and retrodisplacement, require a major operation. I prefer to do this work at the regular operative hour, on a patient who has been suitably prepared for operation, and not at the time when she has finished labor. If cervicitis is the only complaint this would not itself require an operation, as it can be treated better at some later period by cautery or coagulation current. If it is simply a relaxed perineum the repair of this can be very easily done at the completion of labor in any case.

A SIMPLE METHOD OF TREATMENT OF PROSTATIC OBSTRUCTION: CASE REPORTS*

By J. B. Neil, M.D.,
Knoxville, Tennessee

In 1926, I treated six cases of prostatic obstruction with electrocoagulation, using McCarthy's cysto-urethroscope and Bugbee's electrode. I obtained good results with very little hemorrhage or slough. But the tediousness of the method, combined with the fact that American urologists criticised it severely, caused me to discontinue its use.

After hearing the epoch-making paper of Dr. T. M. Davis and seeing his moving pictures in the spring of 1931, at Memphis, my interest in transurethral methods for the relief of prostatic obstruction was renewed. I read extensively on the subject, visited Dr. Davis at Charlotte, North Carolina, saw his beautiful work, and was convinced that prostatic resection could be safely done by him. His five years' experience, however, had given him a deftness which I, a beginner, despaired of gaining.

I was financially unable to buy the equipment then, and therefore I had plenty of time to hear of the embarrassment of others, who were contending with primary and secondary hemorrhage, infection, chills and fever, epididymitis, embolism, cutting of the external sphincter and bladder wall, incontinence, necrosis, gangrene, toxemia and occasional death. I knew Dr. Davis had been severely criticised on his early statistics, but seeing his work impressed me greatly, and I kept trying to discover some reason for his early success, when he was working with a small instrument incapable of the removal of much tissue as compared with the instruments in present use. I believed other factors must enter into his relief of obstruction. These, I reasoned, were slough and dehydration of tissues. I also felt that since most prostates are infected, a cut into this infected tissue, especially a clean sharp cut, would very probably break down nature's wall and open avenues for absorption into the blood stream, whereas coagulation would seal the lymphatics and prevent this absorption. Hoffmeister points out that there is no absorption into the blood stream following electrocoagulation, because of the occlusion of the lymph vessels. Von Seeman and Schan seem to have proven this experimentally.

Further study of the pathology of prostatic hypertrophy convinced me of the efficacy of the method I shall describe. Since 1910, when Edwin Beer first employed fulguration for the destruction of benign papilloma of the bladder, we have been destroying rather large bladder tumors by this method. It is reasonable to believe that if one can destroy tumors in the bladder one can destroy tumors at the neck of the bladder by this method.

Bugbee, who reported sixty-nine cases treated in 1911, 1913 and 1914, gave as the advantages of this method good visualization, no hemorrhage and no incapacitation of the patient. The disadvantages were: inability to destroy suf-

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ficient tissue with the numerous treatments necessary to destroy the entire growth. Most of his cases came to prostatectomy later. I wrote six leading urologists asking their advice about equipment and the results of the method. The trouble that they were having and the terrific mortality were rather appalling. If experts report so much trouble, what could I expect, being only a novice? But in May, 1932, I bought the equipment. Dr. E. P. Nicely, physician to the Knox County Home for Indigents, agreed to cooperate with me in gaining experience.

We chose an old man who seemed a good risk. His blood chemistry was within normal limits. He had a median bar obstruction with six ounces of residual urine. After an indwelling catheter had been left in place for four days, operation was undertaken. Anesthesia consisted of three tablets of a mixture of allylisopropyl barbituric acid and aminopyrine (allonal), ¼ grain of morphine, and 1/150 grain of atropine by hypodermic thirty minutes before operation; and a caudal injection of 50 c. c. of 1 per cent novocaine. After waiting about thirty minutes, I introduced the McCarthy visualized prostatic electrotome into his bladder. My first cut proved that the anesthesia was a failure. He nearly jumped off the table. As the institution is at a distance from town, no other anesthesia was available. I removed the telescope and substituted for the wire cutting electrode the roller electrode sent with the instrument, not for treatment but for hemorrhage control. I turned on the high frequency generator full power and told Dr. Nicely that I was going to experiment on the patient. I rolled the bar carefully. This took only a few minutes and he complained of very little pain. Following this procedure there was no hemorrhage. A definite trough had been made in the bar. I left an indwelling catheter in place for three days, and on its removal he urinated normally. At the end of two weeks I examined his bladder neck with the cysto-urethroscope. With the instrument set at the verumontanum, I could see clearly into his bladder, whereas before, with the instrument so set, the median bar completely obstructed the view. With the exception of slight bleeding about the tenth day he had made a complete recovery, with no reaction of any kind. He had not even been confined to bed. So far as I know no one has used exactly this technic in handling prostatic hypertrophy.

In the January, 1933, issue of the *American Journal of Surgery*, Dr. Tom Kerwin tells of using a roller with low heat that ranges in high frequency below six milliampere minutes, whereas I use at least sixteen milliampere minutes, or the same amount of current used with the cutting wire loop. Let me go over the action of the high frequency current on the tissues, which most of you understand. When two tin foil plates of exactly the same size are placed one on the back and one on the front of the chest, the greatest concentration of heat is generated half way between them. This is called medical diathermy and is used in the treatment of pneumonia. By reducing the size of the front plate, the greatest concentration of heat is brought nearer this plate. When reduced to a point, all the heat is generated on the point or active electrode. This is called fulguration or surgical diathermy. By regulating the size of this small or active electrode, the greatest concentration of heat can be put at any desired depth in the tissues. As I said before, Beer, Bugbee and Stevens, of New York, used diathermy in 1917, also Luys, of Paris, and other surgeons whom I could mention.

The first cutting current was developed for Dr. Collins, of New York, in 1926. The higher powered generators were developed later, early in 1931, I believe, and were designated surgical units. Our minds seemed to have been centered on the cutting current's cutting, with no thought of using this powerful high frequency current in any other way. If the roller electrode (the size of which seems to be correct from my results) is used with the McCarthy visualized prostatic electrotome cutting current, and the same power which is used with the wire loop, an arc is formed in the tissues and a certain amount of tissue is dissolved and consumed. Beyond the area actually destroyed by this electric arc, sufficient heat is generated to cause dehydration of the adjacent glandular structure. Dubois Raymond observed in his experiments that heat coagulates muscle plasma and brings about an acid condition in the tissues. It was discovered in 1859 that the beginning of death of any tissue is marked by a progressive acidity and subsequent coagulation of plasma. The great amount of heat generated in the prostate gland by the cutting current brings about this acid condition and consequently causes shrinkage and dehydration. The acid condition produced in the prostate would also account for the absence of postoperative pain. This lack of pain is also possibly explained by the coagulation of the small terminal nerve endings. The normal prostate is 88 per cent water. To demonstrate the operation of prostatectomy on the cadaver is almost impossible, because the prostate is shrunken from dehydration. Pathologists tell us that a large percentage of prostates contain cystic areas which also make dehydration easy. I have not been unmindful of the hazards that I might encounter in this method of treatment. I was aware of McCarthy's statement that deep coagulation would be followed by slough, secondary hemorrhage, necrosis and gangrene of tissue, toxemia and death. Since none of these

complications has occurred in my cases, I am strengthened in my belief that my method is dehydration rather than deep coagulation. My experience proves to me that there is far less slough from the roller on high powered cutting current than there is with the roller on coagulating current. I observe, too, that there is very little danger of doing too much with the roller on cutting current. For instance, when the current is first applied there is definite shrinkage up to a certain point, beyond which point there is no visable shrinkage at the time. I believe this shrinkage of tissue occurs in the congested, edematous tumefaction of the mucous membrane. Of course the shrinkage varies with the consistency of the tissues. Some glands are very soft and spongy, the roller sinks into the tissues, and so much of it adheres that the roller must be cleansed before proceeding. This is the type that we are told not to resect. But they respond beautifully to heat. This is reasonable, since the boggy tissue contains more water and the shrinkage is greater. This shrinkage continues for five or six weeks. My first cases, I treated again at the end of three weeks, when residual urine indicated. But in testing a patient who had twenty-six ounces of residual urine before treatment, I found he had six ounces after waiting three weeks, and none after five weeks.

Shrinkage without destruction of tissue was described in a resection brief sent out by Wappler & Company. They were describing low heat. My method gives a double action of the heat. There is the destruction of superficial tissue caused by the electric arc. The intensity of the heat fades gradually into the tissues and in so doing causes the non-destructive shrinkage in the deeper underlying glandular structure. I have had almost no charring and the scar tissue is soft and pliable. This method has all the advantages mentioned by Bugbee and others: good visualization, lack of hemorrhage and no incapacitation of the patient, with none of the disadvantages of Bugbee's method. I have used it in sixty-five cases which have required an average of one and one-half treatments to rid them of residual urine. My patients are carefully prepared for this operation, just as for prostatectomy or resection. I should like to stress the preoperative care. In my opinion at the present time patients do not undergo a long enough preparation for prostatic resection. I believe it is best to insert an indwelling catheter for several days, even before making a urethroscopic examination. I have seen patients who were thrown into urethral chill and more or less shock from urethroscopic examination. At the present time I do not urethroscope any prostatic until he has become accustomed to the indwelling catheter and his bladder infection has cleared up to a certain extent.

Any man attempting this operation should be an experienced urethroscopist and know well his anatomy, physiology, and pathology. One may do as much damage with the roller electrode as with the wire loop if any tissue other than the encroaching prostatic lobes be rolled. In median bar cases, I have confined my treatment to the median line with the idea of conserving Bell's muscles of the trigone, thereby hoping it would regain its normal function. Dr. Hugh Young, in a paper published in 1918, described the function of the trigone, saying that it contracts, shortens, and broadens, and in so doing pulls open the weaker arcuate muscle of the internal sphincter. This was later proven by Wesson's work on physiology of the trigone and vesicle neck. Since we feel that the action of the trigone is essential to the normal act of micturition, everything in our power should be done to conserve the action of this muscle. In suprapubic prostatectomy the internal sphincter is frequently destroyed (depending on the pathological condition present) and occasionally the trigone is put out of commission.

In the resection of median bars, where the entire bar is removed, the trigone, including the muscles of Bell, is always cut. Hence the act of urination is greatly interfered with. Since the high frequency current has an affinity for pathological tissue, the application of heat as previously described will cause a shrinkage of glandular tissue without destroying the muscles of Bell. A normal function of the trigone results.

In conclusion, with this simple method of treatment at hand, I want to urge early diagnosis and treatment. To quote from Randall: nowhere in the current literature does one find an answer to the question, why residual urine? Before the days of prostatic resection, a prostatectomy was advised for any man with a constant residual urine of over four ounces. Consequently, residual urine under four ounces was considered of no consequence. In examining a great many men with the urethroscope, I have found median bars and hypertrophy of the trigone, with no residual urine. I believe that as long as the trigone is capable of pulling open the arcuate muscle of the internal sphincter and

the detrusor muscle is capable of forcing the urine over the dam caused by the bar obstruction, there is no residual urine. It is only after the trigone muscle has failed and the detrusor or bladder muscle has begun to fail that there is residual urine. Of course this increases as the muscles grow weaker. The earliest symptom of bladder neck obstruction or encroachment is frequency of urination. This should not be overlooked.

SUMMARY

(1) An original method with cutting current (high amperage) and roller electrode is described. There is actual destruction or dissolution of superficial tissue by the electric arc, and dehydration of adjacent underlying glandular tissue. The dehydration without destruction is much greater than the tissue actually consumed or destroyed.

(2) The advantages are: there is no mortality. It is a simple office procedure in most cases. There is perfect visualization, there is no hemorrhage, the patient is not incapacitated, and there is very little reaction. There are no complications. Sufficient tissue can be removed, and there are economic advantages.

(3) The possible disadvantages are slough and secondary hemorrhage.

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DISCUSSION (*Abstract*)

Dr. George R. Livermore, Memphis, Tenn.—I heard Dr. Neil read a paper on this subject at a meeting of the Tennessee State Medical Association, and we tried out his method in the City Hospital in our department in quite a number of cases. I do not remember the exact number now, but so far our results have been far from satisfactory. In fact, our results have been practically nihil. I was rather enthusiastic when I heard his paper, for the simple reason that years ago Bugbee used fulguration. I attempted to do the same thing with Collins' electrotome in cases of hypertrophy of the prostate of patients who would not submit to operation. Instead of using a roller, I used the ball electrode. I was able to produce a definite slough. Some patients did fairly well for a few months, and one as long as two years, but after a time all symptoms returned.

Now that we have prostatic resection, which is satisfactory, particularly in the type of patient who will not submit to prostatectomy, and who is too poor a risk for the major operation, I feel that we should stick to it till something is proved to be better.

Dr. A. I. Dodson, Richmond, Va.—At a time when many rapid changes are taking place in the treatment of prostatic obstruction and when excellent results are being obtained by methods which just a few years ago were considered not only useless, but also unwise, we are willing to listen with interest to any new procedure which offers possibilities for further advancement in transurethral work.

I have had no experience with the procedure discussed in Dr. Neil's paper and have been entirely satisfied with resection in those cases that seemed to me amenable to any type of transurethral surgery. Dr. Neil's method seems to offer additional help in the treatment of the patients that may be considered too debilitated for resection. It seems to me that in doing this work as an office procedure, he departs rather radically from our usual teaching. It has been my impression and practice that any instrumental work done on elderly men had best be done in the hospital.

OVARIAN PREGNANCY*

REPORT OF A CASE PROBABLY TUBO-OVARIAN, WITH FULLY GROWN FETUS

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The term ovarian pregnancy implies gestation in the substance of the ovary. While the possibility of its occurrence has been disputed, study of early specimens by different observers seems to have established the fact that the ovum may in rare cases be fertilized before its escape from the graafian follicle and may be developed in the substance of the ovary, constituting a primary ovarian pregnancy. The criteria upon which many gynecologists insist before accept-

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ing a case as one of primary pregnancy are quite rigid. As postulated by Spiegelberg in 1878, they are:

(1) The tube on the affected side must be intact and distinct from the sac.

(2) The fetal sac must occupy the position of the ovary.

(3) It must be connected with the uterus by the utero-ovarian ligament.

(4) Ovarian tissue must be found in the sac wall.

Williams insists that ovarian tissue must be found in several portions of the sac wall.

Fulfilling these criteria, only about 45 instances of undoubted ovarian pregnancy have been reported, and ovarian must be regarded as the rarest form of ectopic pregnancy.

Then there are cases in which the ovum is fertilized after escape from the graafian follicle, and develops on the surface of the ovary encroaching on the ovarian tissue or becoming reimplanted in a ruptured graafian follicle. This is the secondary type and differentiation of it from the primary form, may in certain cases be very difficult. Frank has stated that the "early cases alone can be used in deciding the mode of nidation," and Leo Brady thinks "It may well be that our criteria in deciding what specimens are really examples of ovarian pregnancy have become too rigid."

In 1917, I reported a case of profuse hemorrhage from the ovary as a case of ruptured graafian follicle cyst. The patient had gone a few days over her menstrual period and her symptoms in every respect were those of ruptured tubal pregnancy. But the bleeding was found to be not from a rent in the tube, but from a rent in the ovary. A number of such cases have been reported and in most of them as in my case the pathologico-anatomical examination was not complete.

Heijl states that in cases of this kind (ovarian hemorrhage) which were subjected to careful examination in serial section by competent pathologists, about one-half were shown to be cases of pregnancy. Frossner, in 1916, described a case of early ovarian pregnancy which was proven to be such only after a careful serial examination of the specimen showed the presence of a single chorionic villus, and came to the conclusion that pregnancy in such cases of ovarian hemorrhage could be excluded only by a very careful examination, and probably not even then with certainty. Most of the cases of ovarian pregnancy reported in the literature are early cases, although several writers, including Lau, Stoeckel and Ficte, state that an ovarian pregnancy, especially an intra-follicular one, is more likely to carry to term than tubal or other forms of ectopic. This is explained by the more resistant ovarian cortex and the good blood supply which the ovary affords to the placental circulation.

In the case here reported, the baby was fully developed and as movements were active until nearly nine months from the estimated time of conception, could probably have been saved by prompt delivery at that time.

After the death of the fetus at about the time delivery was due, the mother continued in good health and was delivered by laparotomy nearly six months later and more than fourteen months after the estimated conception. The gestation sac was intact at the time of delivery and at operation presented the appearance of the pregnant uterus at term.

CASE REPORT

The patient was a farmer's wife, aged 29. Her family history is unimportant. She had been married eight years, had had gonorrhea, and two abortions.

On November 15, 1931, she menstruated normally. The period in December was missed. In January and February she had scanty periods, then missed until November 15, 1932. The abdomen increased in size as usual in pregnancy and while the patient could not remember the exact date of quickening, movements were felt for several months. On October 3, 1932, the patient had quite a severe pain in the lower abdomen and thought labor had started. Dr. B. L. Elam, of Centralia, Oklahoma, who saw her at that time, gave her two hypodermic injections of morphine on successive days. He stated that while the pain was present, a body which he took to be the head would rise up in the right iliac region. After this attack of pain no more movements were felt. On November 15, 1932, she menstruated, as stated, and again at the regular time in December, January and February, 1933. On November 25, 1932, she came to our office. At this time the abdomen was enlarged to the extent of a full term pregnancy. The cervix was small, long and rather high. No movements had been felt for seven weeks and no fetal heart sounds could be heard. Our diagnosis at the time was death of the fetus with missed labor. We suggested the giving of quinine and castor oil with the hope of bringing on labor. This was done, but with no result. The patient felt well, was able to do her housework and menstruated regularly.

We heard nothing more from her until April 4, 1933. The condition at this time was practically the same as it was at the time of our last examination in the previous November. An x-ray was taken at this time, which showed the bones of the fetus and confirmed the diagnosis of pregnancy.

Delivery by abdominal section was advised and done the following day under spinal anesthesia.

The appearance presented at operation was exactly the same as that of a pregnant uterus. The wall of the sac on incision was as thick as the wall of the

uterus ordinarily is and only after delivery of the child, when it was noted that the wall of the supposed uterus did not contract as usual, did we suspect that the pregnancy was extra-uterine. It was then found that the sac was on the left side of the uterus in the position of the left ovary, the uterus being small and in such close contact with the sac as to be difficult to recognize. The mass, including the uterus, was removed. The left ureter was dilated to the size of a lead pencil and was in intimate contact with the sac.

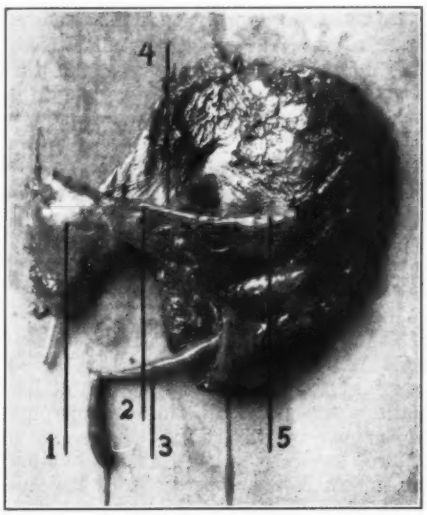

Fig. 1
1. Uterus. 2. Tube. 3. Cord. 4. Ovarian ligament. 5. Fimbriated end.

The patient made a smooth recovery and left the hospital in two weeks.

EXAMINATION OF THE SPECIMEN

The baby, a male, weighing 7¼ pounds, was surprisingly well preserved. The mass of the sac occupied the position of the left ovary and the ovarian ligament is distinctly seen connecting the mass with the uterus. The left Fallopian tube could be traced over the surface of the sac its entire length. The fimbriated end was somewhat adherent to the sac and this is the one thing that makes primary ovarian nidation questionable. Dr. Ralph L. Thompson, of St. Louis, who examined the specimen, found ovarian tissue in the sac over an area about one by three inches. He says:

"At the part I have marked there is a layer of ovarian tissue forming the wall to which there is placental attachment (nidation). So to this extent at least there is ovarian pregnancy. I cannot definitely say that this is not tubo-ovarian, as the ovarian tissue is about where the end of the tube should be."

It seems very probable that a tubal abortion occurred, depositing the fertilized ovum either on the surface of the ovary or into a ruptured graafian follicle.

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SYMPOSIUM ON MALARIA—Part 1 ‡

SOME RESEARCH PROBLEMS IN MALARIA*

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In endeavoring to select a fitting subject for your Chairman's Address it occurred to me that perhaps a short discussion of some research problems connected with malaria, problems which have, as yet, received but little attention, might prove of interest to the Committee. Ten years ago, had most physicians been asked the status of our knowledge regarding yellow fever, the answer would undoubtedly have been that the etiology and epidemiology of this disease had been entirely solved with the exception of a few minor details; yet within that period of time the supposed cause of the disease, *Leptospira icteroides,* has been proven non-existent, the organism bearing this name being identical with *Leptospira icterohemorrhagiae* of Weil's disease; other species and even genera of mosquitoes than *Aedes aegypti* have been proven experimentally capable of transmitting the infection; the contagious nature of yellow fever under certain conditions has been incontrovertibly proven; and many other important and new facts regarding the virus of this disease have been contributed by the various research boards maintained by the International Health Division of the Rockefeller Foundation and by others.

It appears to me that the situation of our knowledge regarding malaria is quite similar today to that of our knowledge of yellow fever a decade ago. We are prone, I believe, to regard our present knowledge as practically complete and we speak very glibly of the life cycles of the malaria plasmodia, the species of these parasites, and the factors concerned in their transmission, forgetting that much that we believe to be fact is based upon very little experimental evidence and that recent work has shown that much still remains unknown or unproven as regards the etiology and epidemiology of the malarial infections.

It is my purpose in this address to call attention to some of the problems still unsolved in regard to these infections in the hope that a brief discussion of them may stimulate research which may add greatly to our knowledge of malaria.

The Species of Malaria Plasmodia.—The first problem to be discussed is the question of the existence of species of malaria plasmodia other than the three classical species generally accepted, namely: *Plasmodium vivax, Plasmodium malariae,* and *Plasmodium falciparum.*

Within the past year the work of James, Nicol and Shute[1] (1932) has demonstrated that *Plasmodium ovale,* observed and named by Stephens,[2] in 1922, is a distinct species of human malaria plasmodium, differing in morphology both in man and in the mosquito vector from the three species mentioned above. This plasmodium, which was first described by myself[3] in 1900, but not named, has been passed through fourteen paretic patients and transmitted to four malaria-free individuals by the bites of infected *Anopheles quadrimaculatus* by the observers mentioned and they say regarding its specific status:

> "In the meantime we can say definitely that there remains no doubt that *P. ovale* is a separate species and that its morphological characters in both the human and insect host are characteristic and constant."

I have recently reviewed the nomenclature of this species[4] (1933) and those interested are referred to that review, but it appears certain that we must accept this species, and thus *P. ovale* becomes the fourth species of malaria plasmodium demonstrated as parasitic in man.

As is well known, still other species of human malaria plasmodia have been described, but not generally accepted, and it is believed that a most careful and extended research should be made regarding the morphology of all malaria plasmodia parasitic in man before concluding that we know all that is to be known regarding the species which may occur in man. When it is remembered that expert protozoologists overlooked no less than four or five species of avian malaria plasmodia for generations, including them all in the species known as *Plasmodium*

*Chairman's Address, National Malaria Committee (Conference on Malaria), meeting conjointly with Southern Medical Association, Twenty-Seventh Annual Meeting, Richmond, Virginia, November 14-17, 1933.

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‡EDITOR'S NOTE.—The Symposium on Malaria, papers and discussions from the National Malaria Committee, meeting conjointly with the Southern Medical Association at Richmond, Virginia, November 14-17, 1933, which are to be published in the SOUTHERN MEDICAL JOURNAL, will appear in three parts: Part 1 in this issue, Part 2 in June, and Part 3 in July.

praecox, it would certainly not be surprising if malariologists may also have overlooked or confused species of human malaria plasmodia.

The discovery by Napier and Campbell[5] (1932) of a species of malaria plasmodia in monkeys which has been shown by Sinton and Mulligan[6] (1932) to have a cycle of schizogony of only 24 hours is most interesting, as it allies this species with that described by Marchiafava and Bignami and others as a quotidian species of estivo-autumnal plasmodium, which I have regarded as a subspecies of *Plasmodium falciparum*. The work of James and Kauntze[7] (1930) and of Garnham[8] (1933) renders still more necessary a careful morphological and biological study of the plasmodia causing estivo-autumnal malaria. James and Kauntze, as a result of their researches, state that it is uncertain whether the estivo-autumnal plasmodium of Kenya Colony, in East Africa, agrees in morphology with *P. falciparum*, while Garnham, in a later study of this plasmodium, concludes that it differs markedly in morphology from the classical *P. falciparum*.

All of these observations should stimulate research in the morphology of the malaria plasmodia both in man and in the mosquito vectors, for it is undoubtedly true that this phase of malarial research has been much neglected in the past, largely owing to the acceptance of the three classical species of human plasmodia and the dictum of the "old masters" that no other species exist.

The Malaria Plasmodia of Man and Monkeys.—A second unsolved problem in malaria is the relationship, if any, between the plasmodia of man and monkeys. Several species of malaria plasmodia have been described in monkeys, apes and chimpanzees, and in these animals occur species that are morphologically identical with the three generally accepted species of man, as well as species that differ from these in morphology. The question of the possible identity of these species with those parasitic in man has not been answered, although most suggestive data have recently been added to our knowledge of the subject.

In 1931, Clark and Dunn[9] endeavored to transmit the malaria plasmodium of the red spider monkey (*Ateles geoffroyi Kuhl*) of Panama to human volunteers by direct inoculation of the blood of monkeys containing the plasmodia and by the bites of mosquitoes infected with it. The direct inoculation intravenously or subcutaneously of blood containing the monkey plasmodium into seven human volunteers was followed by negative results, but in one of eight human volunteers bitten by mosquitoes infected with the plasmodium an irregular temperature occurred for a few days and one or two bodies thought to be plasmodia were observed in the blood. The authors considered their results as showing that this plasmodium is not identical with any of the human malaria plasmodia.

The first undoubtedly successful experiments in transmitting monkey malaria to man are those of Knowles and Das Gupta,[10] reported in 1932. These observers were successful in transmitting a malarial plasmodium found in *Macacus rhesus*, a common Indian monkey, to three human volunteers by subcutaneous inoculation of the blood of monkeys containing the plasmodium. In all of these volunteers, fever and other symptoms of malarial infection developed and numerous plasmodia were found in their blood agreeing in morphology with those occurring in the monkeys and differing in morphology from the human plasmodia, although somewhat resembling *P. malariae* of man.

These observations demonstrate that at least one species of plasmodium causing malaria in monkeys may be transmitted to man under experimental conditions and indicate the necessity of continued research in this direction. If it should finally be demonstrated that some of the species of malaria plasmodia observed in monkeys are identical with those parasitic in man many puzzling features of the epidemiology of malaria in certain apparently uninhabited or sparsely settled regions would be explained and much could be added to our knowledge of the biology of the malaria plasmodia of both man and monkeys. A most valuable and interesting review of the malaria plasmodia which have been described as parasitic in the lower monkeys has recently been published by Sinton and Mulligan[11] and those interested are referred to that contribution.

With the exception of one very doubtful experiment, that of Mesnil and Roubaud,[12] in 1920, who claimed to have transmitted *P. vivax* to a chimpanzee, none of the malaria plasmodia of man have been transmitted to monkeys or any other animal. However, the number of experiments along this line have been very limited and much more work should be done upon the subject before the statement is justified that human malaria cannot be transmitted to monkeys or any of the lower animals.

The Life Cycles of the Malaria Plasmodia.—There are still unsolved problems connected with the life cycle of the human malaria plasmodia in man. Among these, perhaps the most important are the problems of the cause of long-term relapses and the origin of the gametocytes.

While most of us are content to accept the theory of Ross that relapses in malaria are due to a sudden increase in the number of plasmodia that have been reproducing in small numbers during the quiescent periods, there is still much uncertainty as to the explanation of relapses occurring several months or even a year or more after the initial infection. That such relapses do occur has been proven beyond question, and it is in such cases that it is almost impossible to believe that the theory of Ross is a sufficient explanation. Forms of the human plasmodia differing in morphology from either the schizonts or gametocytes have been described in the blood of relapsing patients by S. P. James[13] (1913) and myself[14] (1906), which have been interpreted as possibly the forms which give rise to long-term relapses. These forms do occur in the blood in relapsing cases and have been confirmed by numerous observers, but opinions regarding their interpretation differ and more careful and extended research as to their significance is needed. Regarding the causation of long-term relapses James, Nicol and Shute[15] (1927) say:

"A reasonable explanation of a recrudescence (return of fever and parasites within 8 weeks after recovery) is that not all the parasites in the red blood corpuscles have been killed or have died a natural death, but this explanation is not reasonable for a relapse which suddenly occurs without warning 8 months after a primary infection which may have been so mild as to be unobserved."

The origin of the gametocytes demands further study before we can consider the subject closed. The universally held opinion that the gametocytes originate from certain of the merozoites that have become sexually differentiated after several divisions of the schizonts is, after all, only an opinion, and is not supported by actual scientific evidence. This statement may seem to be a very extreme one, but careful consideration of the subject will convince anyone that, so far as evidence goes, this explanation of the origin of the gametocyte may be easily controverted. One may as justly claim that sexually differentiated forms are inoculated by the infected mosquito and that after a period of growth they divide and result in the forms which we know as gametocytes. Even if our present conception of the origin of the gametocytes is correct, we are yet ignorant of many of the factors having to do with gametocyte production and the reason why in one individual numerous gametocytes are produced, while in another gametocytes are never present or occur in exceedingly small numbers. It is undoubtedly true that the solution of these problems, as suggested by Knowles, Senior White and Das Gupta[16] (1930), will require the cooperation of the protozoologist and the biochemist.

Another problem that demands attention is the possibility that *Plasmodium falciparum* may reproduce by binary fission during its early stage of development, as claimed by LeDantec[17] (1929), Alessandrini[18] (1933) and others. It is undoubtedly true that one very frequently observes "ring-forms" of this plasmodium showing what might easily be interpreted as binary division of the chromatin dot of the nucleus and these forms are certainly more numerous in pernicious cases of malaria than in the usual infection. Do these forms really indicate binary fission of these young forms of the parasite or what is their real significance? This question remains to be answered and should stimulate research in the subject.

Transmission of the Malaria Plasmodia.—One would gather from the literature that little remains to be discovered regarding the method of transmission of malaria but, as a matter of fact, much remains to be done upon this subject as our knowledge is still very incomplete in certain respects. Among these may be mentioned the following: the relation of mosquitoes other than the anopheline to the transmission of malaria; the possible mechanical transmission of malaria by mosquitoes or other biting insects; and the relations of temperature and humidity to the transmission of these infections.

I think that we all accept the dictum that the malarial infections are transmitted only by mosquitoes belonging to the genus Anopheles, but this dictum is founded upon a surprisingly small amount of experimental evidence, as one can count upon the fingers of one hand the really worth-while attempts that have been made to transmit these infections by mosquitoes belonging to other genera. We were just as sure a few years ago that yellow fever could be transmitted only by *Aedes aegypti*, but today we know that experimentally, at least, many other mosquitoes may transmit the disease and infections have been secured with mosquitoes belonging not only to the genus Aedes, but to

several other genera, including Mansonia and Culex. Our exact knowledge regarding this phase of malaria is no more extensive than was our knowledge of yellow fever when we believed that *Aedes aegypti* was the sole transmitter, and a very great deal of work remains to be accomplished before we can be sure that the anopheline mosquitoes are the only transmitters of malarial infections.

While there are a very few records of attempts to transmit malaria by the bites of mosquitoes other than the anophelines, I have been unable to find in the literature any records of a study of such mosquitoes relative to the possibility of the development of the malaria plasmodia in species other than those included in the genus Anopheles, with one exception. Wenyon[19] (1930), in a discussion before the Royal Society of Tropical Medicine and Hygiene, stated that a Russian worker had shown that the initial stages of development and formation of the oökinete occurred in species of Culex, Aedes and Taeniarynchus, but that further development did not occur. This single observation is certainly insufficient to prove that development never occurs in these mosquitoes, and while I personally believe that malaria can be transmitted only by mosquitoes belonging to the genus Anopheles, I must admit that my belief is based upon very little experimental evidence, although supported by what at present appears to be conclusive epidemiological evidence. From the practical standpoint it is demonstrated that the anopheline mosquitoes are the ones concerned in the transmission of malaria and that malaria disappears when these mosquitoes are eradicated, but it has not yet been proven that mosquitoes belonging to other genera may not experimentally transmit malaria under certain conditions for the reason that very little experimental work has been done upon this subject. Further research may result in additions to our knowledge and would, at least, settle the question of the possible transmission of these infections by mosquitoes other than the anophelines.

Whether or not mechanical transmission of malaria by mosquitoes or other biting insects ever occurs is still an unsolved problem in the epidemiology of these infections. By mechanical transmission is meant the direct inoculation of man with malaria plasmodia which may be present upon, or in, the proboscis of mosquitoes which have been interrupted while biting an infected individual and within a limited time bite another individual. This method of transmission may occur in several infections, notably in human trypanosomiasis, and the observations of Ashburn and myself[20] (1907) and Simmons, St. John and Reynolds[21] (1931) have shown that mechanical transmission of the virus is possible in dengue fever by the bites of mosquitoes belonging to the genera Culex and Aedes. As regards malarial infections little has been done in an endeavor to solve this problem. Mayne[22] (1928) in a series of experiments by means of interrupted feedings of mosquitoes from an infected man to uninfected human volunteers, obtained negative results, using *Anopheles quadrimaculatus* in four individuals and *Aedes thibaulti* in eight individuals. He found malaria plasmodia in the proboscis of both species of mosquito when examined after biting the infected human host and prior to biting the uninfected volunteers, but not after biting the first uninfected volunteer. He believes that unless a mosquito almost immediately regurgitated its stomach contents into the abraded skin it is not likely that infection would occur owing to the rapid death of the plasmodia by drying. Suggestive as these experiments of Mayne may be, it must be admitted that much more work should be done upon this problem before we can be sure that the mechanical transmission of the malarial infections by mosquitoes or other biting insects is impossible. It has been shown that infection with malaria may occur in very susceptible individuals through the inoculation of very small amounts of infective blood and the recent demonstration of the transmission of malaria through syringe needles used by drug addicts is suggestive in this respect. Such mechanical transmission would explain some of the extensive epidemics of malaria in which the unusually rapid spread of the infection to very large numbers of people has been difficult to understand if the cycle of development within the mosquito is absolutely necessary for natural infection. It seems to be conceivable that, given an unusual number of active cases of malaria, unusual numbers of mosquitoes, and a susceptible population, mechanical transmission might have much to do with the origin and spread of malarial epidemics.

Immunity in Malaria.—There are many unsolved problems relating to immunity in malaria and these are more and more attracting the attention of research workers. The recent contributions of Taliaferro, James and others have added much to our knowledge of this phase of malariology, but many questions regarding immunity still remain unanswered. In a recent

publication, Mulligan and Sinton[23] (1933) review the subject of what has been accomplished in the study of immunity and conclude that there is evidence that various strains of the same species of malaria plasmodium differ in virulence; that the virulence of a single strain may be changed by frequent passage through either the mosquito or man; that strains may vary in their response to medical measures or in their power to infect man; that chronic or latent infections, caused by *P. vivax* or *P. falciparum*, and allowed to run their course produce a complete or almost complete immunity to the same strain of plasmodium, but not to different strains, whether infection is attempted through mosquito or direct inoculation; and that there is little, if any, evidence of species immunity even after chronic or latent infections.

All of these conclusions regarding immunity in malaria open up a wide field for research and indicate the need of further work in this fascinating phase of malariology.

I might mention many other unsolved problems in malaria research, but enough have been mentioned to indicate that more work is required before we can be satisfied with our knowledge of some of the most generally accepted data regarding the etiology and epidemiology of these infections. To some it may appear that I have been too radical in questioning certain of these data, but it is believed that where a generally accepted theory or apparent fact is based upon insufficient scientific evidence it is the duty of investigators to supply that evidence, if possible, or at least to admit the inadequacy of the evidence and preserve an open mind upon the subject, and if this address will help to stimulate research along the lines indicated, it will have accomplished the purpose for which it was prepared.

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RECENT WORK IN THE EPIDEMIOLOGY OF MALARIA*

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Last year a report was presented to you by this Committee covering some of the advances made in the study of the epidemiology of malaria. That report was limited to work done outside the continental United States because you were familiar with what was being done in this country and because many of the American aspects of the subject were presented at that meeting. This report is likewise limited, and for the same reason. Attention, however, should again be called to the studies of Boyd and Stratman-Thomas at Tallahassee, Florida, and of Bruce Mayne at Columbia, South Carolina, on induced malaria; and to the studies being made by the Bureau of Malaria Investigation of the United States Public Health Service and a number of the state and local health officers of the southern United States. These studies are of great importance in the epidemiology and control of malaria in the United States.

This report is obviously incomplete, since it was impossible to secure access to or to review all of the epidemiological work on malaria published since the early part of 1932. Apology is offered for these omissions.

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TROPICAL AMERICA

It is noted with regret that the Annual Report of the Medical Department of the United Fruit Company was not published in 1932. The investigations by the medical staff of this Company have added much to the knowledge of malaria, and it is hoped that the results of these studies will again be made available.

In Brazil, the *Anopheles gambiae* (*costalis*), reported by R. C. Shannon,[1] does not seem to have spread from the regions of Natal, where it was discovered.

In British Guiana, Giorgio Giglioli,[2] after nine years of epidemiological investigation of malaria on the Demerara River, reports that quartan malaria is rare in that region, and, when not treated, causes chronic nephritis. This was especially true in children. This disease was found to recur with a striking frequency in certain households as the result of house infection with *P. malariae*. He also reports a study of 61 cases of blackwater fever, in which he found that these cases were proportionate to the new or recent malaria infections, and not to those that were chronic. The epidemiology of blackwater fever was, in many respects, identical with that of quartan malaria. Both of these diseases he believes due to house infection, and he stresses the point that, in house malaria, it is the same strain that is transmitted. In the absence of adequate parasitological knowledge, and on the basis of the clinical and epidemiological characteristics of the disease, he believes with Nocht that blackwater fever is due to infection with certain strains or biological varieties of malaria parasites, morphologically not distinguishable from the ordinary forms; but which, by their biological properties, produce hemolysis. Later, from his studies, Giglioli[3] concludes that definite and specific immunity to hemoglobinuria does exist, and that, as in malaria, it is a relative or partial immunity, a condition of tolerance or premunition, and that it may be racial or acquired. This, he explains, is not immunity to infection, but to its clinical manifestations. These clinically negative unrecognizable infections, which he believes are not rare in certain tropical regions, serve as the reservoir or source of the disease.

From Jamaica, B. E. Washburn[4] reports a severe epidemic of malaria caused by a great increase in the breeding of *A. albimanus*. This increase was precipitated by blocking sea water from a large swamp area and by flooding rains which caused permanent saline ponds to become partly fresh. Breeding decreased and the epidemic subsided when the channel was opened and sea water readmitted to the swamp area.

In Panama, Paul S. Carley[5] concludes that splenomegaly as a sign of malaria is more constant in the mestizo (of mixed white, Indian, and negro blood) than in the negro. One-third of the negro cases and one-fourth of the mestizos had parasites without splenomegaly. The second year's observations on malaria in some insanitated Chagres River villages made by H. C. Clark, Director of the Gorgas Memorial Laboratory, Panama City, Panama, and W. H. W. Komp, Sanitary Engineer, United States Public Health Service, Ancon, Canal Zone, have already been read before this Committee (November 18, 1932). These studies are being continued and will later be reported to you.

In Porto Rico, W. C. Earle[6] made observations on the obstruction to wind by screens. He reports that metal screens of 16 mesh reduce the wind velocity from one-third to one-half. Cloth screens gave very bad results, lowering the wind velocity about four-fifths.

EUROPE

In Europe, a further study has been made of the different races of *Anopheles maculipennis* and their relation to the distribution of malaria and the occurrence of anophelism without malaria. Missiroli, Hackett, and Martini[7] have divided *A. maculipennis* into at least four varieties; namely, *messeae, maculipennis, atroparvus,* and *labranchiae*. They also consider *Anopheles elutus* a well marked race of *A. maculipennis*. By extensive geographical study in Italy and Germany, the distribution of these races has been localized, and each race linked with the hydrological conditions of its larval habitat. They state that all races of *A. maculipennis* are found associated with animals more than with man. Endemic malaria was found only where *A. maculipennis elutus* and *A. maculipennis labranchiae* were abundant and in close association with man. Other varieties, especially *A. maculipennis maculipennis* and *A. maculipennis messeae*, have far less contact with man, and, therefore, are not good natural vectors. These varieties can, however, under certain conditions (scarcity of stabled domestic animals, excessive number of Anopheles, certain weather conditions, and the like) maintain endemic or epidemic malaria. Where these conditions exist, benign tertian malaria of low intensity may oc-

cur; but it rapidly returns to its former level. The distribution of the various races of *A. maculipennis* helps to explain the regular distribution of malaria in Europe.

In Holland, de Buck, Torren, and Swellengrebel[8] confirm the significance of the structure of the egg exochorion for differentiating the anopheline races. They state that it is important to observe the anomalies of blood digestion in the hibernating mosquito after the first meal, and that this should be before the end of December. When blood meals were taken in November and December, they found that long wings require two meals and short wings at least six meals to start ovulation.

In northern Germany, Martini, Mayer, and Weyer[9] found that oviposition of *A. maculipennis* ceases about mid-September. The first eggs of *labranchiae* are laid about the end of March and those of *messeae* about three weeks later.

In Italy, P. H. van Theil[10] separates the anophelines with light gray eggs from those with dark gray eggs on the basis of a study of the intercostal structure. He believes that the variety *labranchiae* must only be used to mean *maculipennis* with light dappled-gray eggs (beamlets on the floats); that *A. maculipennis atroparvus* is identical with the *maculipennis with dark gray eggs* (beamlets of floats absent or practically absent); that, in the future, it will not be necessary to distinguish between the mosquitoes with black and dark-barred eggs, as both belong to *A. maculipennis messeae;* and that *A. maculipennis typicus* is not identical with the variety *messeae*. He confirms the observations of Hackett, Missiroli, and Martini as to the range of the different egg types and their bearing on the presence of malaria. He agrees with Hackett and Missiroli in their explanation of anophelism without malaria. G. Escalar,[11] working in Ardea, reports an experiment in 1932 in which pigs were used as a barrier surrounding a population of 700. The indications were that this barrier served to reduce the spread of malaria.

In Spain, Pittaluga[12] and his co-workers in the delta of the Ebro found no anophelines other than *A. maculipennis maculipennis* and *A. maculipennis atroparvus*. The former was more abundant. With the increase of population and agriculture, even rice fields, the malaria rate dropped. Examination of a large number of the Anopheles showed that 83 per cent contained animal, 9 per cent avian, and 6 per cent human blood. It was observed that, during the period of dispersion in the summer, the females sheltered far from the food supply; but with the approach of hibernation, they remained close to this supply.

G. Zotta,[13] in similar studies in the delta of the Danube, reports another example of anophelism without malaria. The anophelines fed on beasts and birds rather than on man. He reports sporadic cases of malaria, but notes that the disease never spreads.

AFRICA

In Sierra Leone, Davey and Gordon[14] show that the infective density bears a close relationship to the malaria incidence and they define this infective density as the average number of anophelines per room times the per cent of that species that is infective. This infective index has little relationship to the malaria rate among the older age groups because of a high infection rate among them due to reinfection and relapse. They point to the estimation of the density of infective anophelines as a method of calculating the relative risk of inoculation with malaria from the different species or in different localities. Gordon, Hicks, Davey, and Watson[15] report that the malaria vectors at Freetown do not remain in the house after feeding, but that there is a twenty-four hour turnover of the mosquito population. They show that there is no correlation between the number of children (the gametocyte carriers of Africa) and the infection rate of anophelines caught in houses. They found that the amount of daylight illumination of a house was a factor in determining the anopheline concentration, but they suggest that this does not indicate that fewer anophelines enter such houses, but rather that they leave them at an earlier hour.

In Uganda, E. G. Gibbins,[16 17] in his studies, reports that *Anopheles funestus* is the most abundant species, but that *Anopheles gambiae* (*costalis*) is the most dangerous vector of malaria. While six species were incriminated in the spread of malaria, only these two were found in more than one region. He found the naturally infected rate of the *Anopheles* to be as follows: *gambiae,* 18.6 per cent; *funestus,* 14.8 per cent; *theileri* var. *hancocki,* 11.4 per cent; *moucheti,* 8.3 per cent; *marshalli,* 0.3 per cent; and *pharoensis,* 0.6 per cent. The first three named were concerned in the spread of all three forms of human malaria. *A. mauritianus* and *A. transvaalensis* were frequently found in houses, and were observed feeding on man; but they showed

a distinct preference for goat blood, while *gambiae* and *funestus* showed a decided preference for man. The prevalence of males and gambiae and *funestus* in houses appears not to be in relation to the proximity to breeding places. The infestation with these two species seemed to be heavier in houses surrounded by dense vegetation.

ASIA

In Russia, P. S. Djaparidse[18] concludes from his studies of carriers in two districts of Georgia that *P. vivax* results in a mild infection, which may remain in the body for years without clinical disease other than splenomegaly, and that, as the presence of this parasite protects from superinfection with this species, it will, as age increases, be less frequently met with on examination of the peripheral blood. Since *P. falciparum* causes severe clinical disease and leaves the body comparatively soon, almost a complete disappearance of protection against this species is brought about so that, with increasing age, there is an increase of the carriers of this parasite.

In Siam, a study by L. Anigstein[19] reports that, in central Siam about Bangkok, there is little breeding because of silt; but upon stagnation, vast numbers of mosquitoes breed. The Anopheles breeding are largely *barbirostris, hyrcanus,* and *vagus,* with only a few of the malaria vectors, which are *A. fuliginosus* and *A. aconitus.* Malaria is prevalent and severe in the hill section surrounding central Siam. In association with this hyperendemic malaria, *Anopheles listoni, culicifacies,* and *minimus* were found in clear slow-flowing canals. In the hilly section of south Siam, which connects with the Malayan peninsula, he found severe malaria in association with *A. listoni* and *A. minimus;* while along the shore only a moderate amount of malaria was found, and was probably due to *A. ludlowi.*

In China, L. C. Feng[20] noted, during his studies, that maximum malaria occurred in May and September, a month after the rainy months, and was more prevalent near the hills than in the villages more distant from them. *A. minimus,* the most common anopheline, was responsible for an epidemic among imported laborers. He reports that, from epidemiological evidence, *A. maculatus* is believed to be a carrier in Hong Kong; but that no infected specimens have been found in any part of China.

In the Federated Malay States, the studies of A. N. Kingsbury[21] show that *A. maculatus* has the greatest avidity for human blood. No evidence was obtained to show daytime feeding of this species. Some of the other anophelines, *Anopheles hyrcanus* var. *nigerrimus* and var. *sinensis,* which, he states, can be and sometimes are infected with malaria, are not usually of epidemiologic importance, because they prefer blood of cattle and other animals to that of man.

In the Philippine Islands, much epidemiological work has been done by P. F. Russell, R. L. Holt, A. P. West, and A. M. Nono. The studies on experimental epidemiology of avian malaria reported last year have been continued. P. F. Russell[22] found four infected specimens of the *funestus-minimus* sub-group in a catch made from carabaos. He presents strong presumptive evidence to the effect that mosquitoes which had first taken blood from an infected human source were attracted to carabaos for subsequent feeding. Simian malaria and carabao infection are not ruled out, but malaria of carabaos is unknown, and monkeys are very uncommon in Ariato. Russell and Santiago[23] report *A. minimus* breeding in wells in fairly large numbers, and they suggest that, in very dry years, with the disappearance of more favorable breeding places, wells may become of great importance in the breeding of this species.

Some of the studies of P. F. Russell on malaria in the Philippines were presented to you at your previous meeting (November 18, 1932).

In India, many studies of malaria have been and are now being made, the reports of which are published in the Records of the Malaria Survey of India. Covell and Baily[24] made studies in northern Sind immediately before, during, and for eight months following an epidemic. They report that the spleen examination showed that immunity was low. The precipitating cause of the epidemic was excessive monsoon rainfall with flooding and a very high relative humidity. *P. falciparum* was the principal parasite. The peak was reached about the sixth week of the epidemic and was followed in two or three weeks by an abrupt rise in the cres-

cent rate. *A. culicifacies* was abundant at the beginning of the epidemic, but few hatched after the first few weeks. It is believed that active transmission does not take place for longer than six weeks. A year after the epidemic, there was a great increase in the spleen rate with a decrease in the size of the average enlarged spleen.

Manson and Ramsay,[25] working in the Jorhat District, report that *A. minimus* continues to be the only important vector of malaria in Assam. They also studied the bionomics of *A. umbrosus* and *A. ramsayi*, and find that neither seems to be of any sanitary importance. Gupta, Ghanosyam, and Majumdar[26] state that in Assam *A. minimus* breeds in clear water at the grassy edges of hill streams, irrigation canals, ponds, and rice fields, but not in foul water. April showers create temporary breeding places which persist until October.

Knowles and Gupta[27] note that the peak of *A. stephensi* is in July and August, while the peak of primary infections is in October. The peak of crescent production is October to December when the curve of *A. stephensi* is falling very rapidly. They explain that this lack of correlation may partly explain why malaria does not become epidemic in Calcutta City.

A preliminary report is made by Knowles and Gupta,[28] in which they report the experimental transmission of monkey malaria to man. The passage of the parasite through man to the second case appeared to increase its virulence. All three human cases recovered spontaneously.

In Calcutta, Strickland, Roy, and Chaudhuri,[29] working with *A. stephensi*, found that 8 to 41 days were required for gland infection. During the cold months, infection of *stephensi* was easily accomplished; but, during hot weathers and the rains, it was almost impossible. They believe that *stephensi* will not become infected with a temperature above 95° F. or below 62° F., or when the relative humidity is above 85 per cent or below 45 per cent.

Harrison and Ramsay,[30] in reporting a survey of eleven tea estates, report that the intense malaria infection found is due almost entirely to man-made conditions, that is, clearing, draining, pit digging, and the importation of non-immune laborers.

Rice and Savage[31] report that they found *Anopheles philippinensis* naturally infected, and that it probably may be a vector of malaria during the earlier months of the year.

James, Nicol, and Shute,[32] working with a Belgian strain of *Plasmodium ovale*, infected *A. maculipennis* and with these they infected patients. From their studies, they conclude "that there remains no doubt that *P. ovale* is a separate species and that its morphological characters in the human and insect host are characteristic and constant."

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MALARIA MORTALITY IN THE SOUTHERN UNITED STATES FOR THE YEAR 1932*

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As in the two previous years, it has seemed desirable to compile the death rates due to malaria in 1932 for the fourteen Southern states both by states and by counties (or by parishes). The value of this plan becomes increasingly evident as data become available for yearly comparison of the death rate trends. Of the states within the area considered (Virginia, North Carolina, South Carolina, Georgia, Florida, Kentucky, Tennessee, Alabama, Mississippi, Missouri [southern three tiers of counties], Arkansas, Louisiana, Oklahoma and eastern Texas), seven states (Virginia, South Carolina, Kentucky, Tennessee, Alabama, Missouri and Arkansas) have shown a decreased rate over the previous year, five (North Carolina, Florida, Mississippi, Louisiana and Oklahoma) have an increased rate, and two (Georgia and Texas) have shown no essential change. However, on comparing the 1932 rates with those of 1930 it is found that nine states (Virginia, South Carolina, Georgia, Florida, Kentucky, Alabama, Oklahoma, Arkansas and Tennessee) have a net decrease for the biennium, and that in Virginia, South Carolina, Alabama, Tennessee and Arkansas this decrease has been consistent for each of the two years considered. On the other hand, North Carolina, Mississippi and Texas have a net increase for the biennium, although in none of these states, with the possible exception of Mississippi, is there yet an indication of a significant increase. The higher mortality rate in Missouri of 1931 over 1930 has been compensated for in 1932, so that the net result in this State for the biennium is favorable. In Louisiana the 1932 rate is essentially the same as in 1930, in spite of an increase of 1932 over 1931. These data are summarized in *Table 1.*

With an idea of better visualizing the trends of malaria mortality by states within recent years (1925-1932) the rates are graphically represented in the accompanying chart. Arkansas shows the highest consistent rate since 1925, with a peak in 1927, a drop in 1929 to the 1925 status, an appreciable rise in 1930 and a favorable decline for 1931 and 1932, with a net decrease for the eight-year period of 27.6 per cent. Mississippi comes next, with a slight but consistent rise to a peak in 1928, then a marked drop through 1929-1931, and an increase of considerable importance in 1932. (Net decline for period: 25.5 per cent.) Florida comes third with a mountainous rise in 1928 to a peak in 1929 considerably more than twice that of 1925, followed by an appreciable drop during 1930 and 1931, but a slight increase in 1932, with a net increase of 8.6 per cent for the period. Louisiana shows a slight decrease in 1926, a slight increase in 1927 and a consistent decrease through 1931, with a return in 1932 to the 1930 level. (Net reduction: 34.1 per cent.) South Carolina has a gradual increase to a broad peak in 1928-1929, which was more than double that of 1925, then a consistently marked decline to 12.0 in 1932, a figure 15 per cent above the 1925 rate. Georgia has had a mortality rate curve practically paralleling that of South Carolina. (Net increase, 8.4 per cent.) Alabama's curve is similar, except that the 1932 rate (6.7) is 19.2 per cent below the 1925 figure (8.3). Tennessee shows a slight increase up to 1928, since which time there has been a gradual decrease to a rate 33.3 per cent below the 1925 figure. The curve reached its high point for the period (8.4) in 1928 and has since then declined. Oklahoma's rate reached its high point for the period in 1927 (9.4) and since that time has gradually declined except for a slight rise in 1932. (Net reduction: 13.6 per cent.) The Missouri curve, primarily influenced by the high rates in the southeastern counties, has shown a general decrease except for the year 1931, when an increase was registered. (Net decline for the period: 36.7 per cent.) In North Carolina the rate was highest in 1925 (4.3), reached its low point in 1930 (1.4) and since that time has risen slowly to 1.6. (Net decline: 62.8 per cent.) Texas had a gradual rise to a peak of 4.3 in 1929, since which time the rate has been slightly above or below 4.0. (Net increase for the period [1926-1932] 53.8 per cent.) The Kentucky curve has shown no real peak; its highest point was reached in 1926 (2.3), its lowest point in 1932. (Net decline for the period: 16.6 per cent.) Virginia began the period with the lowest state

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Table 1

MALARIA MORTALITY IN 14 SOUTHERN STATES
(Rate per 100,000 population by States)

	1925	1926	1927	1928	1929	1930	1931	1932	1932 vs. 1931	1932 vs. 1930	% + or — for 8-year Period	Counties with Rate Over 20	Counties with Rate Over 50	Net Change of Counties, 1930-31	Net Change of Counties, 1931-32	Net Change of Counties, 1930-32
Arkansas	35.9	38.6	46.4	42.4	36.3	39.6	26.9	26.0	—	—	—27.5	36	9	—37	—9	—39
Mississippi	21.6	21.3	22.0	26.5	15.9	14.0	10.4	16.1	+	+	—25.5	16	3	—23	+31	+7
Florida	13.9	14.8	13.8	25.9	31.3	21.6	13.6	15.2	+	—	+ 8.6	28	9	—18	+9	—10
Louisiana	12.9	10.6	13.3	12.2	9.9	8.4	7.3	8.5	+	±	—34.1	9	2	—14	—1	—6
S. Carolina	10.2	12.2	15.6	22.1	22.0	19.3	13.6	12.0	—	—	+15.0	10	4	—6	—9	—12
Georgia	9.8	9.9	10.2	20.0	23.3	15.2	10.5	10.7	+	—	+ 8.4	38	8	—19	0	—27
Alabama	8.3	8.6	8.3	11.7	16.8	11.3	8.1	6.7	—	—	—19.2	5	0	—19	—11	—26
Tennessee	6.3	6.0	5.9	8.4	6.9	5.4	5.3	4.2	—	—	—33.3	4	0	—3	—9	—12
Oklahoma	4.4	5.5	9.4	7.5	6.8	5.3	3.5	3.8	+	—	—13.6	3	1	—14	+2	—17
S. Missouri	4.9	4.0	3.8	4.3	4.3	3.4	3.9	3.1	—	—	—36.7	6	2	+5	+2	0
N. Carolina	4.3	2.9	1.8	2.2	2.2	1.4	1.5	1.6	+	+	—62.8	1	0	—3	+2	+4
E. Texas	—	2.6	3.2	3.8	4.3	3.7	4.2	4.0	—	+	+53.8	10†	3†	—36	**	**
Kentucky	1.9	2.3	2.2	2.0	1.7	2.1	2.0	1.5	—	—	—16.6	3	0	0	0	—5
Virginia	1.8	1.0	0.9	0.5	0.7	0.6	0.5	0.2	—	—	—88.8	0	0	—4	—5	—8
Total for area	8.0*	8.1	9.0	11.4	10.6	8.7	7.6	6.8	—	—	—15.0	13.5%	3.03%	—15.2%	+0.2%	—13.9%

*Data compiled from 13 states only (Texas excluded because information was not furnished).
**County data not furnished for 1932.
†1931 data; 1932 county data not furnished.
—Indicates decrease in rate.
+Indicates increase in rate.

rate in the group (1.8) and has reduced the rate year by year to the significantly low figure of 0.2 for 1932. (Net decrease for the period: 88.8 per cent.)

In order to appreciate more fully the trends in the several states it is necessary to examine the rates by counties for the year 1932. Arkansas, which still leads the group as the most intensively malarious state, had a reduction of deaths in 33 counties, increase in 24 and *status quo* in 18, as compared with 1931, or a net reduction of 9 counties; this in contrast with a net reduction of 37 counties for the preceding year. However, the year 1932 showed a net reduction of deaths in 39 counties over that of 1930; in other words, slightly more than 50 per cent of the counties showed improvement from 1930 to 1932. No single county showed outstanding progress, although Bradley, Conway, Craighead, Desha, Jackson, Jefferson, Lonoke, Ouachita, Sharp, Union and White Counties had a consistent decline in the rate. There are 36 counties (48 per cent) with a rate above 20, and 9 (12 per cent) with a rate above 50. In Mississippi, 17 counties had a decreased rate, 50 an increased rate, and 15 the same rate compared with 1931, with a net increase of 31 counties; contrasted with a net decrease of 23 counties for the previous year. For the biennium there was a net increase of 7 counties. Marked increases occurred in Adams, Leflore, Washington and Yazoo Counties. Sixteen counties (19.5 per cent) have a rate over 20 and 3 (4 per cent) a rate over 50. Florida also had a net increase of counties with higher mortality rates (decrease, 22; increase, 31; *status quo*, 14; contrasted with 35, 17 and 15, respectively). For the biennium, a net decrease of 10 counties

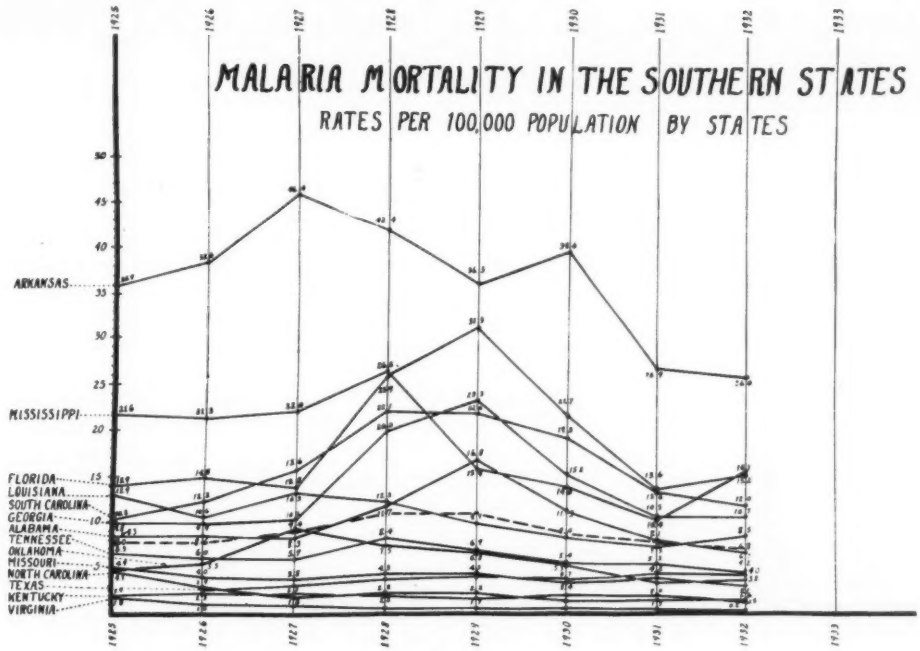

Chart 1

was tabulated. Improvement was particularly evident in Citrus, Dixie and Levy Counties, which lie in the same area; regression in Jefferson and Wakulla Counties, which are situated in the same area. Twenty-eight counties (41.8 per cent) have a rate over 20, and 9 counties (13.4 per cent) have a rate over 50. Louisiana showed marked retardation for the year (decline in 23 parishes, increase in 22, *status quo*, 19; as contrasted with 32, 18 and 14, respectively). The net change for the biennium was a decrease of 6 parishes. All of the increase occurred in the Delta region and near-by parishes north of the Red River. LaSalle, Madison, Tensas and West Carroll Parishes showed significant changes for the worse. However, there are only 9 parishes (14 per cent) with a rate over 20, and only 2 (3.1 per cent) with a rate over 50 (Madison and Tensas Parishes). South Carolina continued improvement during the year, with a net gain of 9 counties, and a net gain of 12 counties for the biennium. Decreased rates were marked in Berkeley and Orangeburg Counties, while Beaufort County increased. Ten counties (21.7 per cent) have a rate over 20 and 4 counties, a rate over 50.

In Georgia no net gain in counties was registered (47 counties decreased, 47 increased and 67 indicated no change). For the biennium the favorable net change was 27 counties. Increases were especially evident in Calhoun and McIntosh Counties, respectively, on the west and east edges of the "wedge" of limestone outcrops, whereas marked decline in the rate was shown in Jenkins County, in the northeastern part of the limestone solution area. Thirty-eight counties (23.6 per cent) have a rate over 20, and 8 (4 per cent) have a rate above 50.

Alabama continued moderate improvement, with a net decrease of 11 counties, contrasted with a net favorable county index of 19 for the preceding year. For the biennium there was a net gain of 26 counties. Progress appeared to be general, rather than confined to particular counties. Only five counties (7.4 per cent) have a rate over 20 and none over 50. Tennessee likewise continued the favorable trend with an increase in the number of negative counties and a net number of 9 counties which

registered a decrease in the rate. Gibson County showed the most decided decline for the biennium. Only four counties (4.2 per cent) have a rate over 20, while none show a rate above 50.

Oklahoma has indicated an unfavorable trend during the year, with a net increase of two counties with a higher rate than in the previous year. Marked increases occurred in Choctaw and McCurtain Counties in the Red River Valley. Three counties (4 per cent) have a rate above 20 and one (1.4 per cent) has a rate above 50. Of the 33 counties in southern Missouri included in this survey there was some indication of recovery from the unfavorable rates which prevailed in 1931. Decreases were registered in Butler, Dunklin and Pemiscot Counties, while increases were indicated in Mississippi, New Madrid and Stoddard Counties. For this group of 33 counties the rate for 1930 was 15.4, increased to 21.0 for 1931, and decreased to 15.7 for 1932. This corresponds exactly with the favorable vs. unfavorable county trends for the biennium. Six counties (18.2 per cent) have a rate over 20 and 2 (6.06 per cent) have a rate above 50. North Carolina shows signs of a slightly unfavorable trend, with a net of two counties having increased rates and an unfavorable index of 4 counties for the biennium. Of the 100 counties in the State only one (Onslow) has a rate above 20 (25.9).

County data from Texas for the year 1932 were not made available. In Kentucky the average trend is favorable, although 3 counties (Fulton, Livingston and McLean) have had increases; on the other hand, a marked decline has been registered for McCracken County, the rate for the biennium having decreased from 30.2 to 2.2. Only three counties (2.5 per cent) have a rate above 20, and none over 50. Finally, Virginia has continued the consistent reduction in the malaria death rate, with a net gain of 5 counties for the year and of 8 counties for the biennium. There are now no counties in the State with a rate above 10.

By way of summary for the Southern states area (see *Table 1*) there has been a 0.2 per cent loss of counties showing decreased malaria mortality rates for 1932 over 1931, although the gain for the biennium is 13.9 per cent. Thirteen and one-half per cent of the counties have a rate over 20 and 3.03 per cent have a rate above 50.

Considering the states in question as a whole, the rate curve for malaria mortality began in 1925 at 8.0, rose slowly to a 1928 high of 11.4 and then receded to the 1932 low of 6.8, a figure 15 per cent lower than in 1925. If these data are a fair basis for comparison, it may be concluded that during this period, in spite of certain as yet unknown unfavorable factors which produced a marked increase in the death rate between the years 1927 and 1930, there was an appreciable net reduction in the rate. There is an indication, however, on the basis of 1932 returns and even more definite in the unofficial incomplete 1933 data, as yet untabulated, that the rate is again rising.

It is, therefore, timely to inquire into the possible conditioning factors which apparently play the important role in producing cyclic excrescences of the malaria mortality curve. Such factors may be hypothecated as intrinsic (that is, such as increased virulence of the parasite itself) or extrinsic (primarily environmental in type). Although our knowledge of the malaria plasmodia (the estivo-autumnal form particularly concerns us) is indeed vague as respects its relation to the human subjects, it seems hardly conceivable that the parasite has any properties which would be expressed in cyclic tendencies of the death rate. On the other hand, floods, poverty and hard times without question are responsible for the following conditions favoring the parasite: (1) lowered nutrition of the patient; (2) his failure to see a physician; (3) his failure to take quinine or other malaricidal drugs; and (4) last, but not least, increased exposure to infection. The answer to this inquiry cannot be reached without extensive study, including a thorough analysis of meteorological data, the relative frequency of the tertian and estivo-autumnal parasites in communities and their correlation with environmental factors by months, years and cycles, as well as the conditions which favor breeding of mosquitoes known to transmit the infections in particular localities. Furthermore, the possible introduction of new strains of plasmodia into a given area must not be forgotten.

With the rate for malaria mortality over 20 in 13.5 per cent of the counties and over 50 in 3.03 per cent of the counties in the southern United States, and with indications of increased mortality for 1933, malaria is a significant disease in this area.

PRELIMINARY OBSERVATIONS ON THE "HIBERNATION" OF *ANOPHELES QUADRIMACULATUS* IN SOUTHERN LOUISIANA*

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In a previous communication the writer (Hinman, 1931) considered the winter breeding and activity of culicine mosquitoes in this region. The present report is an account of observations on the activity of *Anopheles quadrimaculatus* in southern Louisiana made over a period of the past two and a half years. The most important data are from Fort Jackson, a deserted fort on the west bank of the Mississippi River, about 65 miles south of New Orleans. Owing to this distance, frequent or regular visits to the Fort have not been possible, but during the past winter the Fort has been under observation twice monthly.

The possibility of survival of anopheline mosquitoes throughout the winter, in the adult state, is of great importance in the epidemiology of malaria. Mitzmain (1916) in answer to the question "Is mosquito or man the winter carrier of malaria?" concluded that man is the sole winter carrier. However, the presence of considerable numbers of adult *Anopheles* and their activities during the winter months have been reported by Mitzmain (1915, 1916) in Mississippi, Metz (1920) in Florida, Van Dine (1921) in northern Louisiana, Barber, Komp and Hayne (1924) in southern United States, Balfour (1928) in North Carolina and by Boyd and Weathersbee (1929) in North Carolina. Space does not permit a summary of the observations of all these authors, but the general consensus of opinion is that the activities of the adults continue in winter and that there is no true hibernation of any anopheline in the southern United States.

In contrast to the behavior of *Anopheles quadrimaculatus* in the southern United States, Freeborn (1921, 1932) has reported very interesting data on the seasonal life history of its counterpart, *Anopheles maculipennis*, in California. During the late fall, the so-called "prehibernation" forms appear and have a strikingly different habitus, feed frequently, may migrate considerable distances and these migrant forms can and do transmit malaria during the fall and winter. Kligler and Mer (1931) and Kligler (1932) have reported somewhat similar data in the migration of *Anopheles elutus* in Palestine.

THE WINTER BEHAVIOR OF ANOPHELES QUADRIMACULATUS AT FORT JACKSON

Fort Jackson would seem to be an ideal place for the hibernation of adult Culicidae. The Fort is in a very good state of preservation, well protected from winds, on low land with plenty of moisture. It is surrounded by a moat, overgrown with water-hyacinth (*Piaropus crassipes*). With the exception of borrow pits along the levee of the Mississippi River (likewise containing water-hyacinth) there are few or no other suitable breeding grounds for *A. quadrimacultaus*. A narrow strip of land on the west bank of the river is cultivated, chiefly orange groves, but beyond this the country is largely salt marsh, with varying degrees of salinity, excellent breeding grounds for *Anopheles crucians* (coastal variety), *A. atropos*, *Culex salinarius* and *Aedes sollicitans*. The moat and borrow pits, referred to above, do contain *Anopheles quadrimaculatus* larvae, in small numbers, practically the year around. They are, however, permanent bodies of water, containing minnows and other larval predators which may well account for the relatively small numbers of larvae.

The Fort was first visited on February 19, 1931, at which time anophelines (both *quadrimaculatus* and *crucians*) were present, but not in great numbers. Males were observed and some of the females gave evidence of recent emergence. A visit on March 3 revealed little change in conditions except that the anophelines were slightly more active. The Fort was examined once during June, July and August and adult *A. quadrimaculatus* were observed on each occasion. The numbers of these were insignificant when compared with conditions seen on November 27, 1931. At this time, enormous numbers were found. It was estimated that certain rooms of the Fort contained in excess of 10,000 adults per room, largely female *A. quadrimaculatus*. Considering alleys and passageways, there are probably over a hundred rooms in the Fort. Allowing for the fact that there was an uneven distribution of adults throughout the building, there were undoubtedly several

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million anophelines present. The Fort was visited again on December 11 and notes recorded at that time indicate that the adults were even more abundant than at the end of November. Estimates made on hurried counts from small spaces indicated twenty to forty thousand in some of the rooms.

Unfortunately at this time, the true significance of these hordes of female anophelines was not fully appreciated as regards their seasonal life history. The press of other work did not permit another visit until March 19, 1932, when relatively few adult *quadrimaculatus* were observed. The Fort was examined once during April, May, June and July, 1932, and field notes indicate comparatively few adults. On November 7, the Fort was found to contain multitudes of imagines. Accurate observations were not possible owing to the absence of a flashlight, but these were no doubt chiefly females. By the end of December, there were still several million *A. quadrimaculatus* present. Counts in certain areas averaged as high as twenty-five per square foot. The maximum concentrations were in the darker, more moist and more protected parts of the Fort, while the wind-swept portions revealed few adults. There had been some decrease since the first part of November, possibly as great as a half. An unusually cold November combined with a variable temperature throughout December would account for considerable reduction, especially in the more unprotected areas of the Fort. A visit in the middle of January, 1933, showed a further appreciable diminution, but still vast numbers were present. No males were found and the females had little or no evidence of blood meals or marked ovarian development. The majority seemed to be old specimens with possibly a few freshly emerged females. The day was warm and there was some activity of the adults, a few offering to take blood. By February 6 the numbers of *A. quadrimaculatus* were markedly reduced. There was a much greater activity with the females more willing to feed and a number observed outside the Fort. Many of the females within the Fort had blood meals and showed evidence of ovarian development. Few male *quadrimaculatus* were found. It seems evident that some time during the last half of January or first few days of February, a definite dispersion took place. A few of these females laid eggs in near-by breeding places as evidenced by a small number of very young larvae, but probably the majority migrated some distance. Throughout the winter anopheline larvae were present in the moat in various stages, but were very scarce. On February 20, relatively few adult *quadrimaculatus* were present within the Fort. Many of these were active, willing to suck blood and appeared to be freshly emerged. During the previous two weeks, there had been an unusually cold spell of weather. In the latter part of March it was difficult to find any *quadrimaculatus* in the Fort. A few *crucians,* nearly all with blood meals, were present, but male anophelines were rare. By continued dipping a number of small anopheline larvae were obtained, both *quadrimaculatus* and *crucians.*

METEOROLOGICAL CONDITIONS AT FORT JACKSON

The accompanying weather statistics have been compiled by averaging the data given for New Orleans and Port Eads, Louisiana. The latter station is at the mouth of the river, about forty miles from Fort Jackson. Since these two stations are the nearest to the place under consideration, it was believed that data compiled in this way would most closely approximate conditions at the Fort. The temperature variations between these two locations were found to be rather slight, Port Eads having a slightly higher mean temperature. The fluctuation in rainfall was considerable when compared month by month, but annual rainfall revealed but slight differences.

APPROXIMATE METEOROLOGICAL SUMMARY AT FORT JACKSON*

	Precipitation Inches			Max. Temperature Degrees F.			Min. Temperature Degrees F.			Mean Temperature Degrees F.		
	1930-31	1931-32	1932-33	1930-31	1931-32	1932-33	1930-31	1931-32	1932-33	1930-31	1931-32	1932-33
October	5.3	7.0	9.1	88.5	92.0	88.0	47.5	50.5	48.5	72.1	75.0	70.3
November	8.9	2.3	2.6	84.5	85.0	75.5	36.5	45.5	36.0	63.4	69.3	58.0
December	3.4	6.3	4.6	74.5	82.5	78.5	32.5	47.0	30.0	53.4	63.8	59.4
January	4.0	3.9	5.1	71.0	79.5	77.0	32.0	39.0	33.0	52.7	60.8	58.6
February	3.5	3.2	6.8	74.5	81.5	81.0	37.0	43.5	25.0	58.5	65.3	58.3
March	4.1	3.1	6.7	76.0	82.0	81.5	36.0	30.5	39.0	56.8	58.6	62.2

*The writer is indebted to the United States Weather Bureau, New Orleans Station, for meteorological data.

ANOPHELES QUADRIMACULATUS IN OTHER PARTS OF SOUTHERN LOUISIANA

There are certain other old forts not far distant from New Orleans (for example, at the Rigolets, Chef Menteur) which have been visited at various seasons by the writer. The two forts mentioned above are situated at the edge of larger bodies of water and are subject to rather strong winds. Few mosquitoes of any species have been found resting in them during the winter months, but no regular visits have been made. In addition to these buildings, the writer has inspected irregularly, during the past three winters, a number of deserted dwellings, barns, pig styes, hollow trees, and so on, in the vicinity of New Orleans. His observations are more or less in agreement with those of Barber, Komp and Hayne (1924) and other writers referred to above. Adults may on occasion be found abundant during the winter in various resting places which afford good protection. They undoubtedly become active during warm spells and this may lead to oviposition, as evidenced by the presence of very small larvae. Yet some of these females apparently do survive, unfed, for long periods and this condition might be regarded as a "pseudo-hibernation."

DISCUSSION

Unfortunately, the writer does not, as yet, have complete enough information as to the true percentage of males present, the ovarian development and percentage of females with blood meals, larval counts from near-by breeding pools, and so on, to form a definite picture of the seasonal life history of *Anopheles quadrimaculatus* at Fort Jackson. His observations lead him to believe that some time during the early part of November the females begin to collect at the Fort, possibly migrating considerable distances (since there is only a limited amount of breeding in the immediate vicinity), that a fair percentage of these females "hibernate" over a period of from two to three months in a relatively inactive condition, that late in January or early in February dispersal of these females takes place and this distribution may be over a wide area. This dispersion might account for the sudden appearance of many *A. quadrimaculatus* in regions where no larvae were present.

It is difficult to reconcile this hypothesis with the established theory of the winter activity of *quadrimaculatus*. The writer has had a considerable field experience with this species in the vicinity of New Orleans and has found nowhere other than at Fort Jackson evidence of such concentration of females over the long hibernating period. Two theories suggest themselves: (1) that further search will reveal additional places where *quadrimaculatus* assemble in vast numbers and spend a long period of relative inactivity; (2) there is variation within the species, extending all the way from continuous activity throughout the winter to this unusual instance of suspended activity. It seems unlikely that a separate race of this species has been located, although this is possible.

It is to be hoped that a detailed project, on a cooperative basis, may be undertaken in the near future to elicit further information on the winter behavior of this anopheline. This situation would seem to offer ideal conditions for experiments, by means of staining, to determine the length of life of adults during the "hibernating" period, the percentage of survival under given atmospheric conditions, activity and homing habits during winter months, dispersal range, the effect of fumigants, and so on. Such a project would necessitate considerable time and labor.

If additional "hibernating" places of this species were located, it would afford opportunities for control measures against the adults. Fumigation of such places as Fort Jackson should be quite feasible and might exert an appreciable influence on the breeding of the form.

The possibility of malaria transmission by these "hibernating" forms has been considered by Freeborn (1932), Kligler and Mer (1931), and Kligler (1932). Freeborn believes that "late fall and winter transmission of malaria by these migrant forms is possible and actually occurs." Kligler and Mer concluded that mosquitoes destined for hibernation become infected during their first feeds and that they may be responsible for sharp epidemics. The writer has no data available as to the infectivity of these "hibernating" forms. There is no accurate information as to the incidence of malaria in this vicinity, but mortality records and inquiry indicate that it is very slight.

OTHER ANOPHELINES AND CULICINES AT FORT JACKSON

While there are enormous stretches of salt marsh in close proximity to Fort Jackson which breed large numbers of *Anopheles crucians*, but relatively few have ever been found within the Fort. This is especially true during the winter

months. At Buras, a distance of five miles, many *Anopheles atropos* adults may be obtained during the fall, winter, spring and early summer, yet this insect has never been captured within the Fort. Similarly, *Aedes sollicitans* does not seek protection in this building. *Uranotaenia sapphirina* females have been found "hibernating" within the Fort in enormous numbers. More information will be published elsewhere concerning this species.

SUMMARY

A study of the winter activities of *Anopheles quadrimaculatus* at Fort Jackson, Louisiana, indicates that females of this species collect within the Fort in enormous numbers during November, possibly migrating considerable distances, and that a significant percentage of these "hibernate" over a period of two to three months. During the latter part of January or first of February, the females disperse from the Fort. No other quarters have been located where this species congregates in such hordes. The overwintering of large numbers of females is of great importance in the epidemiology of malaria as well as in the survival of this species.

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DISCUSSION (*Abstract*)

Dr. W. V. King, Orlando, Fla.—That adult females of *Anopheles quadrimaculatus* become active during periods of warm weather throughout the winter has been frequently noted and has led to the conclusion that true hibernation does not occur with this species. So far as I am aware, the observations made by Dr. Hinman constitute the first record for the species of what appears to be a marked fall migration under circumstances that strongly suggest their intention of going into hibernation. There are, however, certain similarities shown by the data to the semi-hibernation behavior of *A. maculipennis* in California as recorded by Freeborn and, as is the case with the California species, the individuals gathered in the Fort will undoubtedly prove to be more or less active during periods of warm weather. From the scarcity of host animals, though, it is not likely that more than a very small proportion would be able to obtain blood meals in the immediate vicinity, even if so inclined. It is to be hoped that Dr. Hinman will have opportunities to secure additional information on their daily activities as well as to determine, if possible, the source of such enormous numbers. As mentioned by the author, Fort Jackson lies at the edge of extensive salt marshes in an area where *Anopheles crucians* and *A. atropos* are normally the prevalent anophelines. A very narrow strip of higher land extends along the banks of the river and I am under the impression, from a visit to the area a number of years ago, that fresh water breeding places are extremely limited for a distance of many miles. The migration may, therefore, represent very much longer flights as well as a much larger winter survival of individuals than had previously been realized. Moreover, experiments conducted by myself in New Orleans in 1916 indicate that malaria parasites in infected mosquitoes would readily survive the lowest temperatures encountered at this latitude.

Of further interest in connection with the observation is the contrast shown with the winter habits of *A. quadrimaculatus* in other localities. From personal experience in other parts of Louisiana, especially in the northern section of the State, no indication of a definite fall migration has ever been noted. The species invariably shows a great diminution in numbers during the fall months and, with the advent of cold weather, an almost complete disappearance from such resting places as hollow trees, underneath buildings, and so on. Even those remaining inside of buildings can no longer be found as a rule after a night or two of frosty weather. They reappear during warm spells, take blood meals and develop ova, disappearing again with temperatures below 38 to 40° F. Many hours and days have been spent by members of the Bureau of Entomology in searching for them during these periods Hollow trees and logs have been split open, hay sheds, cotton and corn cribs, accumulations of Spanish moss, and every available crack and crevice have been examined. On one occasion, I believe, Mr. Van Dine even tore down the ceiling and the roof of a building in an attempt to locate their hiding place. Nevertheless, the fact remains that we are still unable to point out the cold weather retreat of *quadrimaculatus* in a region that probably represents a more typical part of its range than the subtropical section below New Orleans.

MALARIA SURVEYS IN FLORIDA*

PRELIMINARY REPORT

By T. H. D. GRIFFITTS, M.D.,†
Jacksonville, Florida

The title of this paper is broad enough to incorporate all phases of investigations to determine the existence of malaria and all factors relating to its prevalence. While some general anopheline surveys have been made and in two counties (Alachua and Bradford) county-wide anopheline (adult) surveys have been conducted, this report has to do largely with malaria blood indexes in the rural white and negro public schools of eight counties in the fall of 1932 and the spring of 1933.

In selecting the counties in Florida in which to establish the parasite indexes eight counties were included, six of which showed death rates during the preceding five-year period of over 100 per 100,000 population. The eight counties were Bradford, Citrus, Dixie, Flagler, Gilchrist, Leon, Levy and Taylor. Flagler is an east coast county; Bradford and Gilchrist are northerly interior counties; Leon northwesterly, and Citrus, Dixie, Levy and Taylor Counties are in the northwesterly Gulf Coast section.

Approximately 90 per cent of the rural school children were included in this parasite survey. Ninety-eight per cent of the pupils in school at the time readily submitted a drop of blood for examination. The standard thick film was made on each case, the Giemsa method of staining used, and the same trained technician examined all of the specimens. In most of these counties, in addition to the examination for malaria parasites and their type, note was made of eosinophilia (that is, eosinophils of more than 4 per cent). Since there has been generally conceded a positive relation between hookworm disease, or infestation, and malaria, we are now making a study with the same blood specimens to determine whether the hookworm case suffers more or suffers less from concomitant malaria. We know, in general, that eosinophilia means intestinal parasites and that the great majority of these are hookworm infestations, at least. It is little added work on the part of the microscopist to report on eosinophilia along with the malaria parasite report. It is our purpose to continue these examinations to include a sufficient number to be of statistical value. It may be stated here, however, that, so far, in the limited number examined there seems to be no greater number of malaria infections among the eosinophilics than in those children not showing an increase in the eosinophils. At the present we do no more than raise the question: "Does hookworm infestation or hookworm disease increase the liability to malaria or does malaria infection make more serious hookworm infestation?" Our data are yet too meager to present.

The Florida malaria parasite index in school children includes the examination of 9,235 white and negro pupils in 136 rural schools (79 white schools with 6,814 pupils and 57 negro schools with 2,421 pupils). Of the 6,814 white children, 260 were found infected, or a rate of 3.8. Of the 2,421 negro children, 234 were found infected, or a rate of 9.7. As to species of parasites, 123 of the 260 positives among the white children were *P. falciparum* (47.1 per cent *P. falciparum*) and 79, or 30.4 per cent, *P. vivax*, the remaining 22.5 per cent being either mixed infections or species undetermined. Of the 234 negroes found positive, 146, or 62.4 per cent, were *P. falciparum*, 54, or 23 per cent, were *P. vivax* infections, with one *P. malariae*, and the remaining 14 per cent mixed infections or species undetermined. These figures indicate a higher rate of *vivax* infections found among negroes than would be expected following the results of experiences of Boyd, Mayne and others who have had difficulty in infecting negroes with *vivax* strains in malaria therapy. The infection rates in the individual schools ran from 84.2, the highest, down to zero (in 41 of the total of 136 schools). In other words, 30 per cent of the schools examined showed no infected pupils by the single thick film examination. This emphasizes the fact that malaria, in the counties of Florida in which indexes have been established, is a series of endemic foci with practical freedom from malaria prevalence in other parts.

In addition to the blood indexes already made and the six or eight other counties with high reported mortality rates in which parasite surveys are contemplated, the Bureau of Vital Statistics has inaugurated a system of checking and verifying reports of deaths from malaria to ascertain the types of malaria causing deaths, and not merely accepting reports of "malaria" as

*Read before National Malaria Committee (Conference on Malaria), meeting conjointly with Southern Medical Association, Twenty-Seventh Annual Meeting, Richmond, Virginia, November 14-17, 1933.

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the cause. Already this checking is bearing fruit and when the physician realizes that more specific data are required, fewer reports of malaria in cases of doubtful or undetermined causes of death are submitted. Given a district or county with relatively high estivo-autumnal malaria, a higher mortality is expected than where benign tertian prevails. By checking blood indexes with reported deaths in the same districts, it is hoped ultimately to establish a more or less direct relationship between reported deaths through the verified certificates and the blood index. As it has been in the past, a reported mortality rate has little relation to the actual prevalence of malaria, but was, of course, indicative of estivo-autumnal malaria or incorrect reporting of causes of death.

The following table presents an analysis in summary of the malaria parasite indexes by counties and schools in eight counties of Florida during the fall of 1932 and the spring of 1933:

MALARIA BLOOD INDEX
Florida Schools, 1932-1933
Thick Films

		Number Examinations	Positive Malaria		E. A.* Per Cent	B. T.* Per Cent	Mixed* Per Cent	Sp.?* Per Cent
			Number	Per Cent				
Bradford County	21 schools	1683	32	1.9	37.5	18.8	3.1	40.6
Citrus County	24 schools	1015	59	5.8	30.5	59.3	0.0	10.2
Dixie County	16 schools	899	41	4.5	70.6	17.1	2.5	4.9
Flagler County	9 schools	473	11	2.3	81.8	9.1	0.0	9.1
Gilchrist County	7 schools	781	19	2.4	26.4	52.6	0.0	21.0
Leon County	9 schools	655	62	9.5	87.1	4.9	1.6	6.4
Levy County	39 schools	2249	213	9.4	60.0	23.9	6.6	9.5
Taylor County	11 schools	1520	56	3.7	39.4	41.8	—	18.8
Grand total	136 schools	9275	493	5.3	54.2	28.4	2.0	15.1

*E.A. = estivo-autumnal; B. T. = benign tertian; Mixed = tertian and estivo-autumnal; and Sp? = rare parasites wherein definite species determination was not feasible.

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PELLAGRA AND PERNICIOUS ANEMIA

An increase of the dietary deficiency diseases has been expected in the lean years of the depression. It is possibly owing to the great development of dietetic principles in the past two decades that morbidity with poverty has not been worse.

Pellagra[1] is classified in text-books as a deficiency disease. It resembles pernicious anemia in many ways and is reported to respond, like pernicious anemia, to liver feeding.[2] The analysis and comparison of foods effective in control of pellagra and pernicious anemia are of interest from an etiological standpoint. Pellagra is apparently closely associated with deficiency of vitamin G, sometimes called the pellagra-preventive factor, which is found in yeast, lean meat, liver and in many other foods. Pellagrins are reported to improve if they receive an abundance of vitamin G. Purified sources of vitamin G do not increase the red blood count and hemoglobin of pernicious anemia. However, according to Castle,[3] of Harvard, all the liver preparations which produce remissions in pernicious anemia contain this vitamin. Castle and his associates have demonstrated that lean beef after incubation with normal gastric juice will stimulate blood formation and remission of pernicious anemia. Lean beef is ineffective before treatment with gastric juice. The Harvard workers have gone further and demonstrated that if heated yeast is incubated with normal stomach juice and administered to patients with pernicious anemia, satisfactory remissions are obtained. Vitamin G plus what Castle calls an intrinsic factor, from normal stomach juice, form a third substance which is hemopoietic in pernicious anemia.

Ruffin and Smith,[2] of Duke University, have studied the effect of vitamin G upon thirty-seven hospitalized patients with typical symptoms of pellagra. The patients were given a very limited basic diet, a diet devised to resemble that on which the disease had developed. It consisted of fat meat, field peas, hominy, cane syrup and lard, to which were added minerals, protein, and concentrated sources of the known vitamins except G. It was desired to see to what extent improvement could be obtained by hospitalization if vitamin G were not supplied. A few of the patients improved markedly on the deficient diet, and their skin lesions healed. However, if the arm or leg of such a patient was exposed for half an hour a day for several days to the direct rays of the sun, dermatitis returned on the exposed surface, often in quite severe form. The tongue and buccal mucosa again became fiery red, and gastro-intestinal symptoms also recurred. If the patient on the G-deficient ration then was protected from the sun for a few days without change of diet, he apparently recovered. Symptoms could be repeatedly evoked by sunning a portion of the body for half-hour periods for a few days. These patients who improved without vitamin G were in a condition which might be described as latent pellagra. They remained well while in the hospital, but on exposure to light showed the results of the deficiency syndrome. An abundance of vitamin G was then added to their

1. Wood, E. J.: Pellagra. McCrae's Osler: Philadelphia: Lea & Febiger, **2**:783, 1927.

2. Ruffin, Julian; and Smith, David T.: Treatment of Pellagra with Certain Preparations of Liver. Amer. Jour. Med. Sci., **187**:512, April, 1934.

3. Strauss, Maurice; and Castle, W. B.: Nature of the Extrinsic Factor of the Deficiency State in Pernicious Anemia and in Related Macrocytic Anemias. New Eng. Jour. Med. & Surg., **207**:55. 1932. Ibid., Etiology and Treatment of Anemia in Pregnancy. Lancet, **1**:1198, 1932.

diet; in a short period all symptoms were relieved and this time the patients on exposure to sunshine developed only a little ordinary sunburn, without recurrence of pellagra.

Various foods were employed by Ruffin and Smith as a source of vitamin G. They preferred an aqueous extract of liver which they gave by mouth. Liver extract 343, a preparation potent in pernicious anemia, was given intramuscularly to a few patients, with negative results.

Etiologically, pellagra and pernicious anemia seem both to have an association with vitamin G deficiency. There are many points of similarity between the two conditions. In pellagra the bilaterally symmetrical skin lesions are characteristic. In pernicious anemia skin lesions are not usually seen. Is it possible that two different syndromes of G-avitaminosis develop, one as pellagra in the agricultural classes or in persons who toil in the sun; the other, pernicious anemia, as a manifestation of the same dietary deficiency in persons whose occupations keep them indoors? In studies of pellagra-like symptoms in animals without vitamin G, the factor of sunlight is not usually mentioned.

A common laboratory technic for the destruction of vitamin G in a mixture of foods is by ultraviolet irradiation.[4] Ultraviolet irradiation of ergosterol produces viosterol, and ultraviolet irradiation of lettuce, of rolled oats and of many other foods is a widely advocated method of increasing the vitamin D content, or the antirachitic potency. Attempts are being made to popularize the use of irradiated milk for infant feeding. According to Thatcher, Sure and Walker,[4] pellagra-like symptoms are produced more rapidly in the albino rat if the casein of a G-deficient diet is irradiated. The work of Ruffin and Smith suggests that pellagrous skin lesions and other symptoms are exaggerated in certain persons by exposure to sunlight. It is conceivable that this exacerbation of symptoms is a result of destruction of a small remaining body store of vitamin G by the ultraviolet ray of the sun.

These investigations are enlightening and stimulating, and, as is usual with good scientific work, they open up vistas for further exploration.

4. Thatcher, H. S.; Sure, Barnett; and Walker, D. J.: Preliminary Report on Pathological Changes in the Albino Rat Suffering from Vitamin G Deficiency. Sou. Med. Jour., **23**:143, 1930.

TOXEMIA OF PREGNANCY

One of the most striking changes of human pregnancy is the sudden appearance in the blood and urine, first, of large quantities of the anterior - pituitary - like, or ovary - stimulating hormone; and later of estrin, the ovarian hormone itself. Prolan is usually readily detected in early pregnancy, and its presence forms the basis of the well-known Aschheim-Zondek and Friedman tests. The excretion of prolan increases to about the second to the fourth month of gestation, after which it gradually diminishes till term.[1] Estrin, the ovarian hormone, is present in the urine in small quantity at the beginning of pregnancy. Its amount rises till delivery.[1]

Smith and Smith,[1] of Brookline, Massachusetts, have made quantitative studies of the sex hormones in the blood and urine of patients with toxemia of late pregnancy. They report in their toxemic cases an increased excretion of prolan amounting to more than sixty times the normal. In many patients, a decrease of urinary and serum estrin was also noted. In other words, they find an overproduction of anterior-pituitary-like hormone and an underproduction of ovarian hormone in the majority of a series of toxemic pregnancies. In a patient who was followed through the entire period of gestation, the prolan of blood and urine rose steadily, while the estrin fell. A month after this abnormality was definitely established, she showed symptoms of toxemia, with albumin in the urine, edema, and elevation of blood pressure.

Estrin is believed to have some inhibitive action upon the formation of prolan. Hence to a toxemic patient with high urinary prolan and low estrin, large quantities of estrin were given by mouth. The clinical condition improved, and the prolan excretion diminished.

Murphy,[2] of the University of Pennsylvania, has observed a high excretion of prolan in an occasional normal pregnancy, in two diabetic pregnancies, and in a patient with a severe cardiac disturbance. The highest reported excre-

1. Smith, G. van S.; and Smith, O. W.: Excessive Gonad Stimulating Hormone and Subnormal Amounts of Estrin in the Toxemias of Late Pregnancy. Amer. Jour. Physiol., **107**:128, Jan., 1934.

2. Murphy, Douglas P.: Excretion of Ovary Stimulating Hormone in the Urine in Pregnancy. Surg., Gyn. & Obst., **56**: 914, 1933.

tions of urinary prolan have probably been those of hydatid mole and chorionepithelioma.

A new endocrine product of predictable effect, that is, an endocrine which can be standardized by biological or chemical means, is likely to be tried by physicians of investigative mind for a dozen morbid states which they do not benefit. The hormone effects are so surprising that it is not difficult to persuade oneself after physiological studies that a particular hormone is a long sought cure. "Cures" of course are developing a hundred times more slowly in this era than are means of diagnosis.

It is of interest that prolan and estrin excretion seem to vary quantitatively in several types of abnormal gestation: that they probably have normal limits of excretion, as have the simpler and better known constituents of the urine, urea, creatinine, nitrogen, chlorine, and so on. Further quantitative studies of the hormones in pregnancy will doubtless be valuable. The qualitative tests as generally used are more than sufficiently troublesome for the average physician. It is probable that until tests are further simplified, routine quantitative hormone studies will not frequently be employed.

GLEANINGS FROM RECENT JOURNALS

Malaria Control.—Probably the only public health activity which has increased during the depression is drainage for malaria control. While the personnel and equipment of state and county health departments were being slashed throughout the country, it was suggested that the unemployed, with no training or machinery, could be used in drainage projects to prevent breeding of mosquitoes. This has proved so popular a measure that, according to Dr. L. L. Williams,[1] of the United States Public Health Service, approximately 100,000 men have been at work for the past five months in malaria control drainage under emergency relief organizations. Malaria control as a permanent part of relief work seems to be well established in most Southern states.

Can One Spirochete Cause Syphilis?—Development of rabbit syphilis following inoculation of a test material into the testes is believed to afford evidence of the presence of the virus in the material used. Failure to infect the animal is considered proof that *Treponema pallidum* was absent from the inoculum. It is usually assumed that a single bacterium of a species is sufficient to bring about an experimental disease.

Thomas and Morgan,[1] of Vanderbilt University, inoculated rabbits with material containing no more than one to six active syphilitic organisms. All the animals remained normal; they showed no evidence of the disease after very careful examination and tests. Apparently, say Thomas and Morgan, an irreducible number of spirochetes must be present before infection can develop. Material with six organisms, they note, would give negative dark field tests. It has been demonstrated, they observe, in the case of another spirochete, that as many as 300 organisms per cu. mm. gave negative dark field findings, although mice could be infected by injection of about sixty of the same bacteria. Dilute suspensions containing about fifteen of these micro-organisms per cu. mm. failed to induce infection.

Thomas and Morgan conclude that injection of one or several of the *Treponemas* does not induce syphilis. A negative tissue transfer experiment, therefore, does not prove complete absence of these organisms. The rabbit's body is apparently able to defend itself against a small number of virulent syphilitic organisms after they have penetrated it.

The Eosinophil Count.—Eosinophilia is supposed to be a defense reaction. Increase of the acid staining cells is noted clinically in asthmas, serum sickness, and in cases of intestinal parasites such as hookworm. It is experimentally produced in various ways, all of which, according to Banerji,[2] of Calcutta, involve disturbance of the acid-base equilibrium. Banerji has produced eosinophilia in rabbits by injecting a solution of carbon dioxide into the veins. The eosinophil count increased from 0 to 2.5 per cent in thirty minutes. If the animals were put into a jar where they breathed carbon dioxide, the count rose in five minutes to 8 per cent.

Defense Reaction of a Lymph Node.—After perfusing the popliteal lymph nodes of normal

1. Williams, L. L.: personal communication.

1. Thomas, C. S.; and Morgan, H. J: Single Cell Inoculation with *T. pallidum*. Jour. Exper. Med., **59**:297, March, 1934.

2. Banerji, Nabajiban: Induction of Eosinophilia in Normal Animals. Amer. Jour. Med. Sci., **186**:689, Nov., 1933.

dogs with solutions of streptococci and also of erythrocytes under pressure, Drinkler, Field and Ward[1] note that these organisms did not reach the blood stream. The lymph from peripheral regions does not reach the blood, they say, until at least a single lymph node has been passed. Normal lymph nodes possess a degree of filtering efficiency so great as to make it fairly certain that in a part which is kept at rest early in an infection, practically no micro-organism would escape the nodes in the line of drainage. The value of rest in the early stages of an infection is thus stressed.

TWENTY-FIVE YEARS AGO

FROM JOURNALS OF 1909

Professor of Physiology, Physical Diagnosis and Clinical Medicine in Mobile Urges Need of Vital Statistics.[2]—"Think of it. Unable to say whether the typhoid death rate of the State is 193 or 93 or 9.3 per 100,000 of poulation. * * * An average of 48.7 per cent of all deaths occurring [in Alabama] are reported. * * * The following letter speaks for itself:

" 'I don't believe there is a man north of the Mason and Dixon line who, if he were ordered down here to live, would not believe he was coming to a sure death from fever in a very short time. Practically all the maps in Europe show our section of the country in yellow and with the additional explanation of "feverish." * * * This organization lost four thousand settlers last year through a western railroad's working on "fever and prejudice."

" 'BENJ. THURLEY,

" 'Secretary, Mobile Chamber of Commerce.'

"* * * Be our health conditions good or be they bad, we must know what they really are."

Medical Economics.[3]—"It is becoming increasingly difficult for the general practitioner to make a living. * * * Adequate business training is needed in the practice of medicine because of (1) the increased demands on would-be medical graduates; (2) the growing competition and constant evolution in medical practice. A medical diploma entails a monetary outlay of at least $2,000 and beyond this a year or more of hospital experience is greatly encouraged. * * * Once in actual work, the practitioner of medicine, more than any other wage earner, is up against hard factors that make for low pay, slow pay and no pay."

Infant Feeding.—Findings of Dr. Joseph E. Winters,[1] Professor of Diseases of Children at the Cornell University Medical College, "are presented in the form of aphorisms."

"Never give water from a bottle to a newborn child—refusal to nurse is certain to ensue. Water may be given from a spoon twice the first day. It must not be given the second. Much water, even from a spoon, prevents vigorous nursing.

"Pasteurization dissolves the organic union of mineral and proteid in milk, and thus fails to present the baby with normally constituted organic food. It is a recourse to palm on a credulous public, milk unfit for food.

"An infant is not endowed with the ability to digest vegetable foods. Barley, dextrinized cereal, malt soup, all adventitious and foreign accessions to modified milk enhance mortality. * * * When women's breasts secrete barley gruel, dextrinized cereal * * * these anomalous and strange feeding whims will be founded on some bottom of reason."

Dispensing of Narcotics.[2]—"At a meeting of the State Board of Pharmacy, a ruling was made that after thirty days the dispensing of drugs containing narcotics shall be allowed only on the prescription of a physician. Dr. T. A. Cheatham, State Drug Inspector, announces that he will vigorously prosecute all violators of the law."

Medical Licensure.[3]—"It is announced that on and after July all applicants for license to practice medicine in the State [Alabama] must be graduates of reputable medical colleges. This leaves only four states, Arkansas, Massachusetts, Mississippi and Tennessee, which continue to examine non-graduates in medicine for license to practice."

Osler.[4]—"Dr. William Osler arrived in the United States from England April 21."

1. Drinkler, Cecil; Field, M. E.; and Ward, H. K.: The Filtering Capacity of Lymph Nodes. Jour. Exper. Med., **59**: 393, April, 1934.

2. Mohr, C. A.: A Plea for the Collection of Vital Statistics in Alabama. Gulf States Jour. Med. & Surg., **14**:274, 1909.

3. Snively, I. Newton: Symposium on the Physician's Ethics and Income. Philadelphia County Medical Society. J.A.M.A., **52**:1533, 1909.

1. Therapeutics. Infant Feeding. J.A.M.A., **52**:1533, 1909.

2. Medical News, Georgia. Idem., p. 1672.

3. Medical News, Alabama. J.A.M.A., **52**:1431, 1909.

4. Idem., Maryland. Idem., p. 1433.

Dangers of Early Transportation.[1]—"Dr. Harry H. Harrison, Jackson (Mississippi), was seriously injured in a runaway accident April 7."

Whiskey Prescriptions.[2]—"The appeal of Dr. Erasmus A. Duncan, Salem, from the decision of the State Board of Health, which revoked his license to practice medicine on account of alleged issuance of prescriptions for whiskey in violation of the law, is said to have been dismissed in the Circuit Court April 3."

1. Medical News, Mississippi. J.A.M.A., **52**:1433, 1909.
2. Idem., Missouri.

Book Reviews

Transactions of the American Proctologic Society. Thirty-Fourth Annual Session. Held in Chicago, Illinois, June 12 and 13, 1933. Dr. Cecil D. Gaston, Editor. Birmingham, Alabama: The American Printing Company, 1933. Cloth, $5.00.

This volume contains a number of items of interest to others than proctologists. Of import is the review of proctologic literature for the year of 1932; six hundred and ninety-eight titles are reported, a number of which are abstracted. The paper of Dr. Harry E. Bacon on the use of the Guskin test offers great possibilities in the early diagnosis of carcinoma. These transactions have been ably edited and should stimulate a greater disposition on the part of the medical profession to inquire into what may occur in this least examined portion of the body.

Treatment in General Practice. By Harry Beckman, M.D., Professor of Pharmacology at Marquette University, School of Medicine, Milwaukee, Wisconsin. Second Edition, revised and entirely reset. 889 pages. Philadelphia and London: W. B. Saunders Company, 1934. Cloth, $10.00.

So numerous are the revisions in this edition that the text is practically a new book. Many deletions have been made in treatments that are no longer used and numerous new agents and methods have been included. Among the new pathological conditions presented are: aspirin poisoning; agranulocytosis; blackwater fever; bronchomycosis; cyanide poisoning; erysipeloid; food allergy; gasoline and kerosene poisoning; hiccup; hyperinsulinism and dysinsulinism; hypothyroidism without myxedema; lead poisoning; lymphogranuloma inguinale; malnutrition; methyl chloride poisoning; oncocerciasis, oriental sore, dermal and mucocutaneous leishmaniasis; gonorrheal prophylaxis in the female; strongyloides infection, tear gas burns, tetany, varicose ulcer and a section on vehicles and incompatibilities. It would hardly seem necessary to add this latter section to a book on treatment. This book fills a great need in its presentation of a complete up-to-date treatment for the general practitioner.

The 1933 Year Book of Pediatrics. Edited by Isaac A. Abt, D.Sc., M.D., Professor of Pediatrics, Northwestern University Medical School, with the collaboration of Arthur F. Abt, B.S., M.D., Associate in Pediatrics, Northwestern University Medical School. 548 pages, illustrated. Chicago, Illinois: Year Book Publishers, 1934. Cloth.

The Year Book of Pediatrics contains abstracts of all the more important procedures and ideas that have been reported during the past year. There are a number of reports of congenital goiter. A method for the quick freezing of breast milk has been devised to preserve breast milk. Its advocates claim the original properties of the milk can be preserved for at least four months. Criticism is made of "normal" height and weight standards. It is felt that the use of a width-length index would be more accurate. Rivers (page 171) summarizes knowledge of virus diseases. This is a valuable addition to any medical library.

Contagious Diseases: What They Are and How to Deal with Them. By W. W. Bauer, B.S., M.S., Director, Bureau of Health and Public Instruction, American Medical Association Member, Editorial Advisory Board of Hygiea, The Health Magazine. 218 pages. New York: Alfred A. Knopf, publisher, 1934. Cloth, $2.00.

This book in a popular style will be of special value to parents, as the author gives a general outline of the germ theory of disease, the methods of disease control and the ways of taking care of patients at home in order to avoid the spread of infection. Such common conditions as measles, mumps, whooping cough, diphtheria, poliomyelitis, small pox, chicken pox, rabies, and colds are discussed in detail.

Aids to Pathological Technique. By David H. Haler, M.D., B.S. (Hons.) Lond., Pathologist, Infants' Hospital, S. W. I; Battersea General Hospital, S. W. II. 187 pages. Baltimore, Maryland: William Wood & Co., 1933. Cloth, $1.50.

The writer covers the entire field of clinical pathology, giving the method of examination that he has found most reliable and efficient. In this manner he has made the book both concise and useful.

The Enlarged Prostate and Prostatic Obstruction. By Kenneth M. Walker, F.R.C.S., M.A., M.B., B.C., Jacksonian Prizeman and Hunterian Professor, Royal College of Surgeons, 1911, 1922, 1924, 1933. 223 pages, illustrated. Second Edition. New York: Oxford University Press, 1933.

The chapters on anatomy and physiology are unchanged. The older methods of operation on the prostate have been augmented by carefully prepared chapters on the newer per-urethral methods. Numerous illustrations, some in color, show the appearance of the prostate when subjected to these procedures. The author has added new chapters on other forms of obstruction, as those caused by malignancy and the mechanism of obstruction. The monograph is well written, complete, and authoritative.

The 1933 Year Book of Urology. Edited by John H. Cunningham, M.D., Associate in Genito-Urinary Surgery, Harvard University Postgraduate School of Medicine. 445 pages, illustrated. Chicago, Illinois: Year Book Publishers, 1934. Cloth.

The abstracted articles show evidence of a broadening of the urologist's view to include a greater knowledge of general medicine. This volume includes many abstracts concerning transurethral operations, bladder tumors, ureteral transplantations, intravenous urography, urinary antiseptics and the treatment and prevention of gonorrhea. An excellent resume of the year's important literature.

The Technic of Preventive Medicine in Europe. By Carl Prausnitz, M.D. (Breslau), M.R.C.S. (Eng.), L.R.C.P. (Lond.), Professor of Hygiene in the University of Breslau. 180 pages, illustrated. London: Oxford University Press, 1933. Cloth.

This book comprises the Heath Clark Lectures for 1932. The writer gathered most of the material for these lectures from visits to the various countries so that it embraces first hand information of the teaching of preventive medicine in France, Germany, Great Britain, Poland, Hungary, Czechoslovakia, Yugoslavia, Spain, Greece and Russia. In addition there is a chapter on the work that is being done by the League of Nations. Contrasts and similarities between the work of the different countries are pointed out. A number of the countries are shown to have made progress which is quite remarkable. There are a number of illustrations. The reports show a generalized interest in public health work. An excellent book for the public health worker who is interested in what other nations are doing in this field.

Bacteriology. For Medical Students and Practitioners. By A. D. Gardner, D.M., F.R.C.S., Fellow of University College, Oxford; Member of Research Staff, Medical Research Council. 276 pages. New York: Oxford University Press, 1933. Cloth, $2.25.

A handbook of bacteriology which presents the salient features of the subject. The nomenclature is of the American Society of Bacteriologists. Numerous references are given to larger works at the end of each chapter. This book is suitable to refresh the memory or for the correlation of the subject when studying for an examination.

Physiological Health. Edited by Jay B. Nash, Chairman, Department of Physical Education. 308 pages. New York: A. S. Barnes & Co., 1933. Cloth, $2.00.

This is the fourth volume of a series and has for its purpose the enumeration of the possible contributions of a well organized physical education curriculum to health. Health is defined as consisting of many different forms as untested health, biological health, functional health, structural health, behavioristic health, numerical health, average health, *Gestalt* health, psychological health, and mental health. The position of each one of these to the whole subject is brought out. This book will be read with interest by those interested in physical education.

Southern Medical News

The American Heart Association will hold its tenth scientific session in Cleveland, Ohio, June 12, at the Cleveland Hotel. Dr. Stewart R. Roberts, Atlanta, Georgia, is President.

The American Association for the Study of Goiter will meet in Cleveland, Ohio, June 7, 8 and 9 at the Wade Park Manor. Dr. R. M. Howard, Oklahoma City, Oklahoma, is President; and Dr. A. G. Payne, Greenville, Mississippi, is a member of the Executive Council.

ALABAMA

The Alabama State Medical Society held its sixty-seventh annual meeting in Birmingham April 17-19. Dr. W. M. Cunningham, Jasper, was elected President; Dr. W. M. Salter, Anniston, First Vice-President (reelected); and Dr. Douglas L. Cannon, Montgomery, Secretary (reelected). The three vacancies on the Board of Censors were filled by the election of Dr. Lloyd Noland, Birmingham; Dr. J. D. Perdue, Mobile; and Dr. George H. Searcy, Tuscaloosa. Dr. James R. Garber, Birmingham, is the retiring President.

The Franklin County Medical Society has elected the following officers for 1934: Dr. T. J. Glasgow, Belgreen, President; Dr. Price Clayton, Bankhead, Vice-President; and Dr. N. F. Underwood, Russellville, Secretary-Treasurer.

Deaths

Dr. LeRoy Boyd, Birmingham, aged 70, died February 21 of angina pectoris and arteriosclerosis.

Dr. Henry Green, Dothan, aged 67, died February 22.

ARKANSAS

Dr. S. A. Thompson, formerly of Stephens, has located at Camden and become associated with Dr. J. B. Jameson for the practice of internal medicine and diagnosis.

Dr. George F. Jackson has been appointed Chief, Department of Dermatology, and Dr. A. C. Kirby, Chief, Department of Pediatrics, of Saint Vincent's Hospital, Little Rock.

Dr. H. King Wade, Hot Springs National Park, is the newly elected President-Elect of the Mid-South Postgraduate Assembly, which held its annual meeting in Memphis, Tennessee, in February; and Dr. Ira Ellis, Monette, was elected Vice-President for Arkansas.

Dr. William R. Hunt, Clarksville, completed fifty years of active practice in March.

The Boone County Medical Society has elected the following officers for 1934: Dr. D. L. Owens, President; Dr. D. E. Evans, Vice-President; and Dr. W. H. Poynor, Secretary-Treasurer; all of Harrison.

Deaths

Dr. Robert Lee Little, Judsonia, aged 68, died February 10 of heart disease.

Dr. John V. Arrington, Blevins, aged 58, died February 2.

DISTRICT OF COLUMBIA

The Georgetown University School of Medicine has announced the following appointments of professors and instructors to the faculty: Dr. Laszlo Detre, Special Lecturer in Immunology; Dr. W. F. R. Phillips, Visiting Professor of Anatomy; Dr. Joseph L. Schwind, Associate Professor of Anatomy; Dr. Philip Lewis Gray, Associate Professor of Pharmacology; and Dr. J. R. Schenken, full-time Instructor in Pathology.

The Kober Lecture, an annual event at the Georgetown University School of Medicine, was delivered in March by Dr. Walter Bradford Cannon, George Higginson Professor of Physiology at Harvard University.

Deaths

Dr. Paul Edgar McNabb, Washington, Major, M. C., U. S. Army, aged 46, died February 24 of progressive hypertensive disease and cerebral hemorrhage.

FLORIDA

The Florida State Medical Society met April 30, May 1 and 2 at Jacksonville for its annual session, with headquarters at the Mayflower Hotel.

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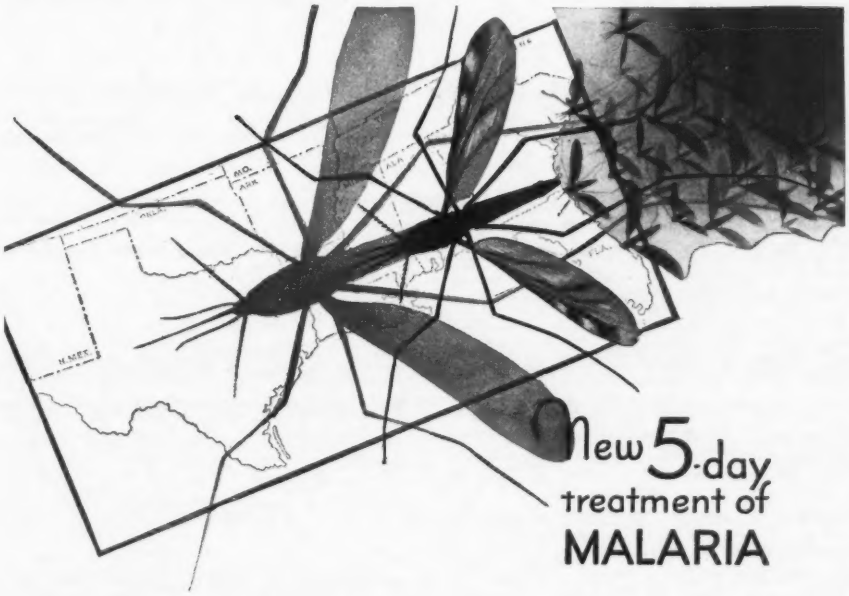

Hitherto: THREE WEEKS' treatment with quinine has been commonly required to eradicate all parasites from the blood of the malarial patient.

Now: FIVE DAYS' administration of Atabrine is sufficient to destroy the schizonts which produce the acute paroxysms and the gametocytes which transmit malaria through the mosquito.

Atabrine has none of the by-effects of quinine which so often interrupt treatment and interfere with the patient's occupation. Relapses are much less likely to occur.

Moreover, Atabrine is economical as only five days' medication is usually necessary in tertian and quartan malaria.

DOSAGE: Adults, one tablet three times daily for 5 successive days. Children, one-half to one tablet, once to thrice daily according to age.

Pamphlet containing detailed instructions will be sent to physicians on request.

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Continued from page 472

Dr. George E. W. Hardy, Tampa, is the newly elected President of the Atlantic Coast Line Surgeons' Association.

Dr. Oliver P. Broadbent, Jacksonville, has moved into new offices in the St. James Building.

Dr. J. S. McEwan, Orlando, was recently reelected a Director of the Gulf Life Insurance Company.

Dr. G. H. Edwards, Orlando, has been elected a member of the Board of Trustees of Rollins College.

Dr. O. G. Kendrick, Tallahassee, is the newly elected Chairman of the Florida Crippled Children's Commission, succeeding Major Charles L. Snyder.

Dr. Paul Kells, formerly an instructor in medicine at the University of Virginia, has moved to Miami and opened offices at 405 North 90th Street.

Dr. G. H. Edwards, Orlando, was recently reelected Chairman of the Orlando Utilities Commission.

The Dade County Medical Society has elected the following officers for 1934: Dr. Roy J. Holmes, President; Dr. F. A. Vogt, Vice-President; Dr. Robert T. Spicer, Secretary; and Dr. H. A. Barge, Treasurer; all of Miami.

The DeSoto-Hardee-Highlands County Medical Society has elected the following officers for 1934: Dr. G. F. Highsmith, Arcadia, President; Dr. Howard V. Weems, Sebring, Vice-President; and Dr. L. W. Martin, Sebring, Secretary-Treasurer.

The Lake County Medical Society has elected the following officers for 1934: Dr. Will Wood, Mt. Dora, President; Dr. R. H. Williams, Eustis, Vice-President; and Dr. W. L. Ashton, Umatilla, Secretary.

The Putnam County Medical Society has elected the following officers for 1934: Dr. A. E. Drexel, President; and Dr. E. W. Warren, Secretary-Treasurer; both of Palatka.

The Seminole County Medical Society has elected the following officers for 1934: Dr. Arthur W. Knox, President; Dr. C. L. Park, Vice-President; and Dr. John T. Denton, Secretary-Treasurer; all of Sanford.

The Volusia County Medical Society has elected the following officers for 1934: Dr. Roy Howe, Daytona Beach, President; Dr. George A. Davis, DeLand, Vice-President; and Dr. Joseph H. Rutter, Daytona Beach, Secretary-Treasurer.

The Walton-Okaloosa County Medical Society has elected the following officers for 1934: Dr. E. P. Webb, Crestview, President; Dr. J. C. McSween, DeFuniak Springs, Vice-President; and Dr. A. G. Williams, Lakewood, Secretary-Treasurer.

Deaths

Dr. Richard Coopender Bankston, Tampa, aged 79, died February 1.

Dr. Edward Temple Willson, Winter Haven, aged 68, died February 21 of coronary sclerosis and arteriosclerosis.

Dr. Birt B. Reeder, Clarksville, aged 54, died March 2 of carcinoma of the stomach.

Dr. James Mitchell Hartley, Hollywood, aged 45, died February 19.

Dr. Cyrus J. Marshall, Sanford, aged 51, died February 19.

GEORGIA

Dr. G. Lombard Kelly, Augusta, has been appointed to succeed Dr. W. L. Moss, resigned, as Dean of the University of Georgia Medical Department.

Dr. Albert Fleming, Folkston, was host at a shad supper in March for members of the Ware County Medical Society.

The Crisp County Medical Society recently sponsored a chest clinic, which was conducted by the Department of Public Health at Cordele.

The Crisp County Medical Society has elected the following officers for 1934: Dr. P. L. Williams, President; Dr. C. E. McArthur, Vice-President; and Dr. J. N. Dorminy, Secretary-Treasurer; all of Cordele.

The Elbert County Medical Society has elected the following

Continued on page 20

PATIENTS BATHE WITH IMPUNITY

● Drybak, the waterproof adhesive plaster, brings a new freedom to patients who must wear strappings or dressings. They can bathe without danger of water loosening the adhesive backcloth. Drybak is sun-tan in color. It stays clean. The edges will not turn up.

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Radiograph showing osteogenic sarcoma of femur.

CASES of bone tumors usually offer only an unfavorable prognosis unless they are detected in their earliest stages. For they metastasize rapidly and ultimately produce fatal conditions.

Each type of bone tumor, whether benign or malignant, requires its own particular measures of treatment and accompanying precautions. But in every case the most important single element in the therapeutic management is a *prompt, accurate, early diagnosis.*

To be sure to detect such tumors at the earliest possible time, refer your patient to your radiologist at the very first suggestion of bone disorder. His radiographs will give you positive information on which to base your diagnosis . . . will permit classification of the tumor . . . will aid in determining treatment.

● *Visit the Eastman Exhibit, Booths 198 and 199, American Medical Association Meeting, June 11-15, 1934, Cleveland Public Auditorium, Cleveland, Ohio.*

Radiographs Provide Diagnostic Facts

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Continued from page 18

officers for 1934: Dr. D. N. Thompson, President; Dr. F. A. Smith, Vice-President; and Dr. A. S. Johnson, Secretary-Treasurer; all of Elberton.

The Ocmulgee Medical Society, Bleckley, Dodge and Pulaski Counties, has elected the following officers for 1934: Dr. W. H. Pirkle, Cochran, President; Dr. J. C. Wall, Eastman, Vice-President; and Dr. I. J. Parkerson, Eastman, Secretary-Treasurer.

The Polk County Medical Society has elected the following officers for 1934: Dr. George M. White, Rockmart, President; Dr. P. O. Chaudron, Cedartown, Vice-President; and Dr. H. R. Perkins, Rockmart, Secretary-Treasurer.

The Tattnall County Medical Society has elected the following officers for 1934: Dr. J. C. Collins, Collins, President; Dr. C. B. Walling, Collins, Vice-President; and Dr. J. M. Hughes, Glenville, Secretary-Treasurer.

Dr. Edgar F. Fincher, Jr., and Miss Helen Nichols, both of Atlanta, were married February 10.

Deaths

Dr. Robert Stephens Reid, Savannah, aged 62, died January 4.

Dr. William Crissey Kellogg, Augusta, aged 59, died February 13 of pneumonia.

Dr. Minot K. Kellogg, Augusta, aged 25, died February 9 of pneumonia.

Dr. William R. Terry, Shellman, aged 71, died February 14.

Dr. John Ledbetter, Eatonton, aged 79, died February 3.

Dr. J. F. J. Hood, Atlanta, aged 65, died February 19.

KENTUCKY

Dr. J. D. Allen, Louisville, formerly associated with the Allen-Sandlin Laboratories, Inc., and the Allen-Sandlin School of Laboratory Technique, Inc., announces the discontinuance of these connections. Dr. Allen will continue to be associated with the Louisville Research Laboratory.

The Knox County Medical Society has elected the following officers for 1934: Dr. Edward Parker, Gray, reelected President; Dr. M. A. Bizzell, Artemus, reelected Vice-President; and Dr. F. R. Burton, Barbourville, Secretary-Treasurer.

The Pike County Medical Society has elected the following officers for 1934: Dr. L. E. Cox, Stone, President; and Dr. M. D. Flanary, Pikeville, Secretary.

Dr. Eugene Joseph Love, Petersburg, and Miss Alberta N. Sheldon were married recently.

Deaths

Dr. Jonas Samuel Gilbert, Allais, aged 59, died suddenly February 9 of heart disease.

Dr. William H. Lester, Dublin, aged 69, died February 16 of pneumonia.

Dr. Benjamin Butler Keys, Murray, aged 53, died January 25 of heart disease.

Dr. Joseph M. Gilbert, English, aged 66, died February 12.

Dr. Algernon Winston Walden, Owingsville, aged 77, died February 28 of cerebral hemorrhage.

Dr. Lewis F. Hammonds, Dunnville, aged 65, died February 21 of cerebral hemorrhage.

LOUISIANA

The Louisiana State Medical Society, at its annual meeting in Shreveport, April 10-12, elected the following officers for the coming year: Dr. C. P. Gray, Sr., Monroe, President-Elect; Dr. Marcy J. Lyons, New Orleans, First Vice-President; Dr. J. M. Gorton, Shreveport, Second Vice-President; and Dr. Paul T. Talbot, New Orleans, reelected Secretary-Treasurer. Dr. Chaille Jamison, New Orleans, is the incoming President.

A chapter of Sigma Xi, of the National Society for the Promotion of Research, was installed at Tulane University of Louisiana in April, the installation ceremonies consisting of the presentation of a charter, a banquet at which Dr. Alton Ochsner was toastmaster, and a public lecture delivered by Dr. E. C. Faust.

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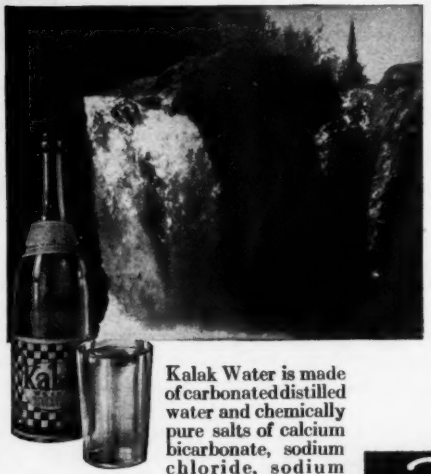
A Pure, Palatable, Carbonated



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Kalak Water is made of carbonated distilled water and chemically pure salts of calcium bicarbonate, sodium chloride, sodium phosphate and bicarbonates of magnesium, potassium and sodium.

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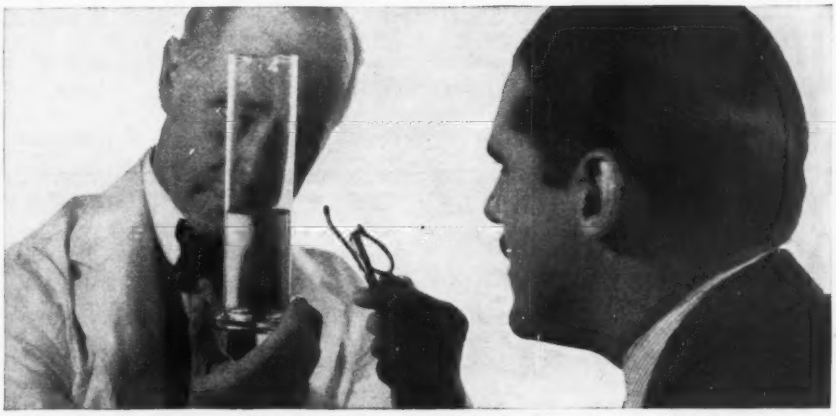

• the result of PYRIDIUM orally administered in urinary infections

The oral administration of Pyridium shortens the period of treatment in the majority of cases of cystitis—pyelitis—urethritis. Pyridium also affords early relief of the distressing symptoms usually associated with urinary infections.

LITERATURE ON REQUEST

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Continued from page 20

Dr. Waldemar R. Metz, New Orleans, President of the Orleans Parish Medical Society, has been chosen to represent the Parish Society on the Directorate of the New Orleans Association of Commerce, at the invitation of the Association of Commerce.

Dr. Charles A. Bahn, New Orleans, President of the Louisiana Society for the Prevention of Blindness, has been chosen a member of the Advisory Committee of the National Society for the Prevention of Blindness.

The Sixth District Medical Society met in Baton Rouge recently and elected the following officers for the coming year: Dr. Sidney Porter, Baton Rouge, President; Dr. F. H. Hanson, Donaldsonville, Vice-President for Ascension Parish; Dr. Charles Voss, Baton Rouge, Vice-President for East Baton Rouge Parish; Dr. E. M. Robard, Jackson, Vice-President for East Feliciana Parish; Dr. A. A. Landry, Plaquemine, Vice-President for Iberville Parish; Dr. Z. J. Gautreau, Albany, Vice-President for Livingston Parish; Dr. L. H. McGehee, Hammond, Vice-President for Tangipahoa Parish; Dr. M. O. Becnel, New Roads, Vice-President for Pointe Coupee Parish; Dr. H. A. Tynes, Grangeville, Vice-President for St. Helena Parish; Dr. John Griffith, Slidell, Vice-President for St. Tammany Parish; Dr. E. E. Lafferty, Bogalusa, Vice-President for Washington Parish; Dr. J. O. St. Dizier, Walls, Vice-President for West Baton Rouge Parish; Dr. C. C. Blakeney, St. Francisville, Vice-President for West Feliciana Parish; and Dr. Cecil Lorio, Baton Rouge, Secretary-Treasurer.

MARYLAND

Dr. Allen Weir Freeman, Baltimore, Professor of Public Health Administration at Johns Hopkins, will become Dean of Johns Hopkins School of Hygiene and Public Health in July, succeeding Dr. Wade Hampton Frost, who will resign upon the expiration of his term. Dr. Frost plans to devote his entire time to his duties as Professor of Epidemiology.

Dr. Adolph H. Schultz, Baltimore, was a guest speaker recently before the Sigma Xi Chapter of Duke University School of Medicine, Durham, North Carolina.

Dr. L. Emmett Holt, Jr., Baltimore, presented a paper at a meeting of the Durham-Orange County Medical Society at Duke Hospital, Durham, North Carolina, recently.

Dr. Calvin Hyman and Mrs. Rose Sagner Sachs, both of Baltimore, were married in March.

Dr. Arthur Karl Koff and Miss Dorothy Hess, both of Baltimore, were married in January.

DEATHS

Dr. Frank William Keating, Owings Mill, aged 64, died February 18 of acute dilatation of the heart.

Dr. William Winter Beall, Baltimore, aged 74, died December 29 of myocardial insufficiency and arteriosclerosis.

Dr. William John Hendry, Baltimore, aged 24, died February 18 of poliomyelitis.

Dr. James Bates Bennett, Baltimore, aged 65, died January 21 of lobar pneumonia and chronic arthritis.

Dr. William A. Frontz, Baltimore, aged 48, died March 23 of heart disease.

MISSISSIPPI

Dr. P. W. Rowland, Oxford, was recently made President of the Mid-South Medical Assembly at its meeting in Memphis, and Dr. Clyde M. Speck, New Albany, was elected Vice-President for Mississippi.

Dr. Hunter Cox has located in his former home, Eudora, to practice medicine.

Dr. D. T. Sayle, formerly of Black Hawk, Carroll County, has moved back to his former home, Highlandale, Leflore County, to practice.

Dr. Ben F. McNeal has moved from Moorhead to Swiftown to practice.

Dr. A. J. McIlwain, formerly of Merigold, has moved to Batesville, and is located in the office of the late Dr. W. C. Lester.

Dr. Lauch Hughes has reopened offices in the Lamar Building, Jackson.

Dr. Katherine McCormick will open offices in Jackson to

Continued on page 24

An Advance in Opiate Medication

Dilaudid

dihydromorphinone hydrochloride

Dilaudid possesses several advantages over morphine for the control of pain and cough. It acts quicker, is a stronger analgesic, and causes fewer undesirable symptoms. 1/20 gr. Dilaudid will usually replace ¼ gr. morphine.

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Oral tablets, rectal suppositories, 1/24 grain.
Ampules, 1/32 grain. Powder, readily soluble.

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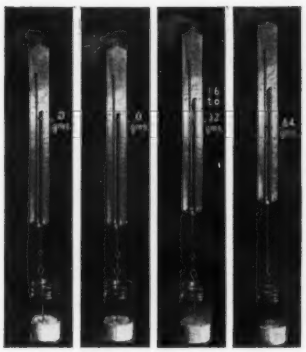

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C—Cow's milk S—Similac
Schematic drawing of the relative size of the curds of cow's milk and Similac vomited by six weeks old puppies after one-half hour's ingestion.

"FAT has a caloric value more than twice that of either carbohydrates or protein and serves very well to make up the necessary energy or caloric requirement. Two of the important vitamins, 'A' and 'D', are associated with the fat of milk and when the diet is low in milk fat these vitamins must be supplied in some other form." [1]

"When milk curdles in the infant's stomach it entangles a large proportion of the milk fat in its meshes and only such fat as lies near the surface of the curd can be reached by the digestive juices. The amount of fat in the curd depends upon the amount of fat in the milk." [2]

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The finer the curd the greater the surface area. The greater the surface area the more exposed are the fats, carbohydrates, proteins and salts to the digestive enzymes. Result . . . a more complete utilization of the food elements.

[1] Marriott: Infant Nutrition, pg. 49.

[2] Talbot: Morse and Talbot, Diseases of Nutrition and Infant feeding, pg. 48.

Samples and literature will be sent on receipt of your prescription blank.

SIMILAC—Made from fresh skim milk (casein modified); with added lactose, salts, milk fat and vegetable and cod liver oils.

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Offices in Most Principal Cities

Continued from page 22

practice obstetrics, after completing her hospital work in New Orleans.

Dr. W. T. Thornhill, formerly of Picayune, connected with the Martin Sanatorium, has moved to Stephenson, and will have associated with him his son, Dr. D. H. Thornhill, who is completing his internship at Touro Infirmary, New Orleans. Drs. Thornhill will look after the medical work for the L. O. Crosby Company.

Dr. W. W. Hickman, formerly of Piave, has moved to Picayune and will fill the vacancy made by Dr. Thornhill at the Martin Sanatorium.

Dr. B. E. Kane has located at Amory.

Dr. Van Dyke Hagaman, Jackson, and Miss Sue Griffith were married in February.

Deaths

Dr. James Samuel Jackson, Belzoni, aged 69, died in October.

Dr. James Andrew King, Forest, aged 60, died December 3 of angina pectoris.

Dr. W. C. Lester, Batesville, aged 47, died recently.

MISSOURI

The Missouri State Medical Society will meet in St. Joseph May 7-10 for its seventy-seventh annual session.

Dr. Joseph Grindon, St. Louis, has been made Chairman of a Centennial Committee appointed by the St. Louis Medical Society to arrange for a celebration of the one hundredth anniversary of the Society, which was planned in 1835. Dr. Charles E. Hyndman, St. Louis, has been made Secretary of the Committee.

The Kansas City Urological Society held a meeting at the Hyde Park Hotel in March.

The Caldwell-Livingston County Medical Society has elected the following officers for 1934: Dr. H. M. Grace, Chillicothe, President; Dr. C. L. Woolsey, Braymer, Vice-President; and Dr. Donald M. Dowell, Chillicothe, Secretary-Treasurer.

Dr. Sylvester Bernard Helwig and Mrs. Hazel Broderick, both of St. Louis, were married in February.

Deaths

Dr. Walter Webster Harrington, Kansas City, aged 54, died February 9 of chronic cholecystitis and uremia.

Dr. Harley S. Norton, Troy, aged 55, died January 19.

Dr. Claude Percival Fryer, Maryville, aged 60, died November 9 of coronary thrombosis.

Dr. Henry A. Brierly, Peculiar, aged 65, died March 1 of coronary thrombosis.

Dr. Noah A. Tesson, Kansas City, aged 70, died February 23.

NORTH CAROLINA

Dr. N. Thomas Ennett, formerly of Richmond, Virginia, has moved to Mount Airy, and is engaged in public health work in Surry County.

Dr. W. S. Rankin, Charlotte, Director of the Hospital Division of the Duke Endowment, recently lectured in the amphitheater of Duke University.

Dr. Adolph H. Schultz, Baltimore, Associate Professor of Physical Anthropology at the Johns Hopkins University School of Medicine, recently made an address before the Duke Chapter of Sigma Xi.

Dr. W. M. Junkin, Murphy, recently Surgeon to the Petrie Hospital, has resigned this position and has moved to Elkins, West Virginia, where he will be a member of the Staff of the Elkins City Hospital.

Dr. Samuel H. Juster has located at Macclesfield since graduating in the 1933 class of the Medical College of Virginia.

Dr. John H. Hamilton, Raleigh, Director of the State Laboratory of Hygiene, recently lectured on County Health Work at Duke University.

Dr. A. C. Thompson, formerly of Asheville, has gone to Washington, District of Columbia.

Dr. A. B. Holmes, Fairmont, has been elected President of the newly organized Robeson County Shrine Club.

Dr. Everett Walker McCauley, Burlington, and Miss Ella Irene Wilson were married in February.

Dr. Bennie Booker Dalton, Red Springs, and Miss Willie Grace Covington were married March 2.

Dr. Joshua Tayloe, Washington, and Miss Mary Ann Jamison were married recently.

Continued on page 26

Prevent Summer Diarrhea

THIS Spring—especially where milk supplies are uncertain—insure with Dryco against Summer diarrhea.

With its higher protein and mineral values, Dryco builds *more* real living tissue—*less* fat and water. It increases resistance to infection and dangerous dehydration. "*Dryco*" *babies do not melt away.*

The low—but ample—fat content of Dryco is another guard against gastro-intestinal upsets. It makes for easy digestibility during convalescence too.

And—remember its safety—*no milk-borne infection has ever been traced to Dryco.*

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Continued from page 24

Dr. Edward Marvin Mann, Mayock, and Miss Ethelyn Anderson Thompson were married recently.

Dr. Jefferson Bivins Helms and Miss Doris McCollum, both of Morganton, were married recently.

Dr. Jeremiah Robert Johnson and Miss Lois Ellis, both of Ramseur, were married recently.

DEATHS

Dr. Richard Elliott Lee, Lincolnton, aged 60, died February 15 of pneumonia.

Dr. Merle Theron Adkins, Durham, aged 58, died February 21 of coronary thrombosis.

Dr. Abram G. Jones, Walnut Cove, aged 89, died January 15.

Dr. George Dillwyn Green, Waynesville, aged 84, died December 18 of chronic myocarditis.

OKLAHOMA

Dr. E. S. Ferguson, Oklahoma City, is the newly elected Chairman of the Oklahoma Section of the American College of Surgeons; Dr. A. W. Pigford, Tulsa, has been elected Secretary; and Dr. LeRoy Long, Oklahoma City, Counselor; the elections having been held at a meeting in Oklahoma City in February.

Dr. E. E. Conner, formerly of Erick, has moved to Mangum and opened offices.

Dr. C. E. Northcutt, Ponca City, is the newly elected President of the Ponca City Chamber of Commerce, and was elected the most useful citizen for the year 1933.

Dr. C. B. Hill, Guthrie, has been appointed to succeed Dr. Robert Allen as County Physician of Logan County.

Dr. McClain Rogers, Clinton, Mayor of his city, was honored recently at a joint meeting of the Rotary and Kiwanis Clubs, and elected the city's most useful citizen for 1933.

Dr. Charles M. Pearce, McAlester, since returning from Memphis, where he attended the Campbell Clinic, has opened offices at 117½ North First Street.

DEATHS

Dr. Abraham Lincoln Blesh, Oklahoma City, aged 68, died February 20 of carcinoma of the lung.

Dr. Harry Calvert Iles, Prague, aged 69, died January 6 of acute nephritis and uremia.

Dr. Jesse Franklin Campbell, Mangum, aged 57, died February 19 of pneumonia.

Dr. Norman Smith Starr, Oklahoma City, aged 45, died January 30.

Dr. John Boasman Rone, Oklahoma City, aged 71, died suddenly February 8 of myocarditis.

Dr. Samuel Erskine Mitchell, Muskogee, aged 61, died February 23.

SOUTH CAROLINA

Dr. Asa Mock Scarborough, Greenville, and Miss Mina Eugenia Chapman were married recently.

Dr. Roy Lee Cashwell, Fountain Inn, and Miss Mary Sullivan West were married in March.

DEATHS

Dr. S. B. W. Courtney, Lake City, aged 58, died February 12 of coronary thrombosis.

Dr. William Clifton Black, Greenville, aged 73, died December 12 of cirrhosis of the liver.

TENNESSEE

Dr. M. K. Moulder has become associated with Dr. S. T. Ross, Nashville, with offices in the Doctors Building.

Dr. A. R. Porter, Jr., formerly of Memphis, has moved to El Paso, Texas.

Dr. J. F. Bradley, Memphis, has moved into new offices at 154 North Cooper Avenue.

Dr. H. G. Bland, formerly of Etowah, has moved to Newport News, Virginia, with offices in the Medical Arts Building.

Dr. Perry Gambill, formerly of Knoxville, has moved to Grey Stone Park, New Jersey.

Dr. G. Madison Roberts, Chattanooga, has moved into new offices in the James Building.

The Henry County Medical Society has elected the following officers for 1934: Dr. A. F. Paschall, Puryear, reelected Presi-

Continued on page 28

Klim message of the month

Your Own Klim Formula Or An Artificial Breast Milk

It has been said that all mixtures used in infant feeding which are imitations of breast milk in respect to the relative proportions of protein, carbohydrate and fat, outwardly seem to affect the infant in a manner similar to breast milk, notwithstanding the fact that they "are entirely lacking in the protective properties which make breast milk the safest and most perfect food for the infant."

(*Kugelmass*)

AUTHORITY: "In designing a formula for the artificially fed infant, what should be the goal? Macy has stated that it should be to surpass breast milk not to equal it. The philosophy that the composition of breast milk offers the best proportions for the artificial formula has given rise to the promulgation of numerous proprietary foods designed to provide a product of chemical composition similar to human milk. It often escapes the attention of the physician that even though the proximate analysis of such food may resemble closely that of human milk, the nature of its ingredients may be very different. The characteristics of milk proteins afford an excellent example. Human milk with a protein content of 1.5 per cent seems to meet the needs of the infant. It does not follow that any milk modification providing equivalent amounts of protein would be equally adequate, unless the protein were chemically, and possibly biologically identical. . . . Because of such intrinsic differences between human milk and its substitutes, the composition of human milk does not offer an optimum pattern for the artificial formula." (Boyd, J. D., *Jour. Pediat.*, Vol. 4, No. 2, Feb. 1934.)

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Continued from page 26

dent; Dr. Elroy Scruggs, Paris, Vice-President; and Dr. R. Graham Fish, Paris, Secretary and Treasurer.

Dr. George Hampton Garmany, Chattanooga, and Miss Alice Harriette Heibeck were married in February.

Dr. Roy M. Lanier, Brownsville, and Miss Mildred Jones were married in February.

DEATHS

Dr. Alvin A. Francis, Knoxville, aged 77, died December 19 of lobar pneumonia.

Dr. Louis Francis Zachary, Livingston, aged 68, died December 1 of heart disease.

Dr. Floyd Patrick Smith, Trenton, aged 39, died February 24 in a Memphis hospital of cerebral hemorrhage.

Dr. Manly Eugene Siler, Mercer, aged 51, died February 12 of heart disease.

Dr. Joseph Lemuel Campbell, Bristol, aged 58, died February 17 of myocardial insufficiency.

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A private maternity home for the care and protection of unfortunate girls during pregnancy and confinement.

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Dr. Paul Edgar McNabb, Major, M. C., U. S. A., a native of Tennessee, aged 46, died February 24 in a Washington, District of Columbia, hospital.

TEXAS

Dr. W. P. Lee, Cisco, was recently reelected City Health Officer by the City Commission for a period of two years.

The following physicians have been elected officers of the Baylor Hospital Staff for the coming year: Dr. R. B. McBride, President; Dr. Tate Miller, Vice-President; and Dr. Edwin L. Rippy, Secretary; all of Dallas.

The Bell County Medical Society has elected the following officers for 1934: Dr. W. J. Graber, Jr., President; Dr. L. B. Leake, Vice-President; and Dr. C. M. Simpson, Secretary-Treasurer; all of Temple.

The Burleson County Medical Society has elected the following officers for 1934: Dr. T. L. Goodnight, Caldwell, President; Dr. G. V. Pazdral, Somerville, Vice-President; and Dr. J. E. Siptak, Caldwell, Secretary-Treasurer.

The Ector-Midland-Martin-Howard-Andrews-Glasscock Counties Medical Society has elected the following officers for 1934: Dr. G. T. Hall, Big Springs, President; Dr. E. V. Headlee, Odessa, Vice-President; and Dr. P. W. Malone, Big Springs, Secretary-Treasurer.

The Harris County Medical Society has elected the following officers for 1934: Dr. Judson L. Taylor, President; Dr. H. J. Ehlers, Vice-President; Dr. M. B. Stokes, Secretary; and Dr. J. Reese Blundell, Treasurer; all of Houston.

The Hidalgo County Medical Society recently had a called meeting for the purpose of reorganizing, the new organization to embrace Starr County, and to be known as the Hidalgo-Starr Counties Medical Society. Officers for the new Society for 1934 will be those already elected for Hidalgo County.

The McLennan County Medical Society has elected the following officers for 1934: Dr. Paul C. Murphey, President; Dr. R. W. Crosthwait, Vice-President; and Dr. C. G. Swift, Jr., reelected Secretary-Treasurer; all of Waco.

The Nacogdoches County Medical Society has elected the following officers for 1934: Dr. T. J. Blackwell, Nacogdoches, President; Dr. C. T. Smith, Nacogdoches, Vice-President; and Dr. Henry Tucker, Secretary-Treasurer.

The Northeast (Fifteenth) District Medical Society has elected the following officers for 1934: Dr. H. R. Smith, Detroit, President; Dr. C. D. Scaff, Clarksville, Vice-President; and Dr. C. A. Smith, Texarkana, reelected Secretary-Treasurer.

Dr. Boyd C. Edwards has moved from Fort Worth to Dodsonville.

Dr. Floyd S. Franklin, formerly of Fort Worth, has moved to New York.

Dr. Charles O. Hook has moved from Fort Worth to San Antonio.

Dr. John M. Hooper has moved from Dallas to Pampa.

Dr. John C. Montgomery has moved from Mineral Wells to Dallas.

Dr. J. C. Perry, formerly of Terrell, has moved to Boston, Massachusetts.

Dr. A. M. Schoor, formerly of San Antonio, has moved to Chicago, Illinois.

Dr. S. B. Slaughter has moved from San Marcos to Crockett.

Dr. Charles Wendelken has moved from Austin to Sinton.

Dr. Temple W. Williams has moved from Wichita Falls to Haskell.

Dr. Henry Redmond, formerly of Corpus Christi, has moved to Los Angeles, California.

Dr. Sidney E. Stout, Fort Worth, and Miss Helene Earldine Robb were married in February.

DEATHS

Dr. Mat Marshall McRee, Dallas, aged 57, died February 26.

Dr. Henry Marcellus Johns, Waco, aged 70, died January 31 of cerebral hemorrhage.

Dr. Thompson Wright Grace, Port Lavaca, aged 67, died November 2 of acute dilatation of the heart.

Dr. John M. Henderson, Waelder, aged 84, died December 14 of typhus fever.

Dr. Francis Marion Bourland, Houston, aged 69, died in February.

Dr. James Dunaway McCann, Raymondville, aged 64, died January 31 of heart disease.

Dr. Joseph W. Jones, Stockdale, aged 70, died January 8.

Dr. A. P. Barclay, Wharton, aged 67, died January 10.

Continued on page 30

"The statement is frequently made that no two babies can be fed in the same manner and that the formula must be individualized in each case. This may be true when certain types of formulas are used, but it is not true in the case of formulas which meet the fundamental requirements. In this connection one should not lose sight of the fact that ***human milk,** which is admittedly the best food for the normal infant, **does not vary appreciably in composition throughout the first year,** the only changes in the feeding of the breast-fed infant being in the total amounts taken and the additional foods which are added to the diet."

Williams McKim Marriott, Infant Nutrition, 151, (1930).

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"The natural food of the infant is ***human milk** characterized by the fact that its quality **changes very little,** the infant's growth being dependent on the increase in the amount of milk secreted."

Julius H. Hess, Feeding and the Nutritional Disorders in Infancy and Childhood, 7, (1930).

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When diluted according to directions, it is **essentially similar to human milk** in percentages of protein, fat, carbohydrates and ash, in chemical constants of the fat and in physical properties.

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Continued from page 28

VIRGINIA

Dr. N. Thomas Ennett, formerly Medical Director of Public Schools, Richmond, has returned to North Carolina, his native state, and has located at Mount Airy, where he is engaged in public health work in Surry County.

The Fauquier County Medical Society and the Loudoun County Medical Society held a joint meeting recently in Warrenton as the guests of Dr. J. Frank Folk and Dr. Martin B. Hiden, both of Warrenton.

Dr. Harvey G. Bland, Richmond, has opened offices in the Medical Arts Building for the practice of surgery.

Dr. William R. Jordan, Richmond, has opened offices in the Medical Arts Building, with practice limited to the treatment of diabetic patients.

Dr. J. A. Grizzard, Drewryville, has been appointed by Dr. R. D. Bates, President of the State Society, to succeed Dr. E. L. Kendig, deceased, as a member of the Committee on Group Societies of the Medical Society of Virginia.

Dr. Allen Southall Lloyd, Norfolk, has opened offices in the Wainwright Building and will specialize in orthopedic work.

Dr. Paul Kells, formerly of the University of Virginia faculty, has located at Miami, Florida.

The Medical Society of Northern Virginia held a called meeting at Winchester recently and formed skeleton county societies for each county holding membership in the Medical Society of Northern Virginia, which was not organized as a county unit. The President, Dr. George Crawford, Strasburg, appointed provisional officers for the various counties as follows: Dr. C. O. Dearmont, White Post, Clarke County; Dr. D. M. Kipps, Front Royal, Warren County; Dr. W. C. Ford, Woodstock, Shenandoah County; Dr. H. H. McGuire, Winchester, Frederick County; Dr. J. G. Brown, Woodville, Rappahannock County; Dr. George Long, Luray, Page County.

The Southside Virginia Medical Association held its one hundred twenty-first quarterly session in March.

The Lee County Medical Society has elected the following officers for 1934: Dr. W. J. Innes, President; and Dr. J. B. Muncy, Secretary; both of Pennington Gap.

Dr. Lucius H. Bracey, South Hill, and Mrs. Gladys Yancey Bramham were married in February.

DEATHS

Dr. Albert Asa Cannaday, Roanoke, aged 70, died February 22 of Ludwig's angina.

Dr. Benjamin H. Swain, Ballston, aged 74, died January 30 of complications following appendicitis.

Dr. James Dallas Kirk, Roanoke, aged 87, died December 22 of pneumonia.

Dr. Elijah Emery Rose, Ewing, aged 80, died December 27 of senility.

Dr. William Penn Reese, Taylor's Store, aged 86, died February 22.

Dr. Robert Daniel Tucker, Powhatan, aged 72, died March 13.

Dr. William Latane Varn, South Hill, aged 43, died March 21 following an illness which resulted from an infection of the hand.

Dr. Clarence Hyde Saunders, Chase City, died March 14.

Dr. Edgar Bentley Noland, Bassett, aged 45, died suddenly March 2.

WEST VIRGINIA

Dr. Roy Ben Miller, Parkersburg, President of the West Virginia State Medical Association, was honored recently with a testimonial dinner which was sponsored by the Woman's Auxiliary.

Dr. W. M. Junkin, formerly of Murphy, North Carolina, has located at Elkins City, where he is associated with the Elkins City Hospital.

The Marshall County Medical Society has elected the following officers for 1934: Dr. C. G. Morgan, President; Dr. R. A. Ashworth, Vice-President; and Dr. J. A. Striebich, Secretary-Treasurer; all of Moundsville.

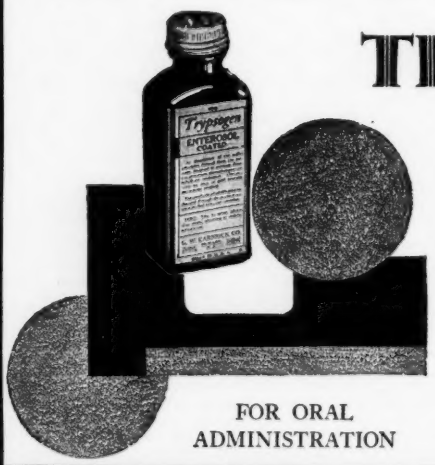
The Raleigh County Medical Society has elected the following officers for 1934: Dr. L. E. Shrewsbury, President; Dr. E. W. Wood, Dr. E. H. Hedrick, Vice-Presidents; and Dr. L. M. Halloran, Secretary-Treasurer; all of Beckley.

DEATHS

Dr. Franklin McKinley, Parkersburg, aged 85, died January 16 of myocarditis and nephritis.

Dr. J. W. Myers, Philippi, aged 61, died January 16 of cerebral hemorrhage.

A COMBINATION of all of the active derivatives of the pancreas which control carbohydrate metabolism together with the specific proteins necessary for regeneration of the pancreatic tissues.



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(Wheatmeal, oatmeal, cornmeal, wheat embryo, alfalfa leaf, and beef bone. Supplies vitamins A, B, E, and G and calcium, phosphorus, iron, copper, and other essential minerals.)

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YOUR own wife can verify that when cereal is cooked one hour, 18 cubic feet of gas are consumed daily or 540 feet monthly. At $1.50 per thousand cubic feet, the gas bill is increased $0.81 per month.

Another reason Pablum is more economical than ordinary cooked cereal is that no allowance must be made "for the pot." The mother can prepare the *exact amount* required. If the child clamors for more Pablum, which frequently happens because of its delicious flavor when properly prepared, another serving of this hot-or-cold cereal can be prepared in less than a minute.

The above economic factors are in addition to the important advantage that because of its quick preparation, the mother is not compelled to arise an hour earlier in the morning to cook cereal over a hot stove. Instead, she can obtain the extra rest which is so necessary for her health after confinement and during nursing.

Pablum, moreover, is rich in minerals and vitamins, is base-forming and nutritious.

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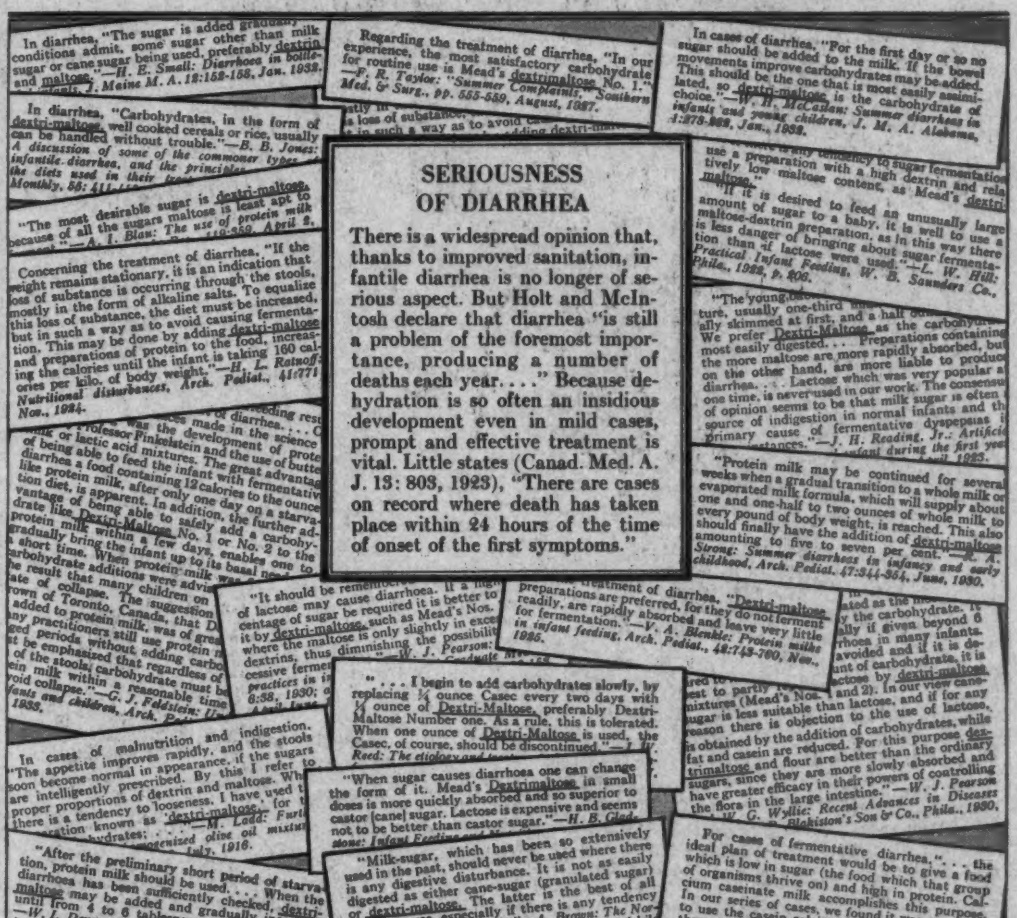
DIARRHEA

"the commonest ailment of infants in the summer months"

(HOLT AND McINTOSH: HOLT'S DISEASES OF INFANCY AND CHILDHOOD, 1933)

One of the outstanding features of DEXTRI-MALTOSE is that it is almost unanimously preferred as the carbohydrate in the management of infantile diarrhea.


SERIOUSNESS OF DIARRHEA
There is a widespread opinion that, thanks to improved sanitation, infantile diarrhea is no longer of serious aspect. But Holt and McIntosh declare that diarrhea "is still a problem of the foremost importance, producing a number of deaths each year. . . ." Because dehydration is so often an insidious development even in mild cases, prompt and effective treatment is vital. Little states (Canad. Med. A. J. 13: 803, 1923), "There are cases on record where death has taken place within 24 hours of the time of onset of the first symptoms."

Just as DEXTRI-MALTOSE is a *carbohydrate* modifier of choice, so is CASEC (calcium caseinate) an accepted *protein* modifier. Casec is of special value during the summer months (1) for colic and loose green stools in breast-fed infants; (2) in fermentative diarrhea in bottle-fed infants, (3) as a prophylactic against diarrhea in infections.

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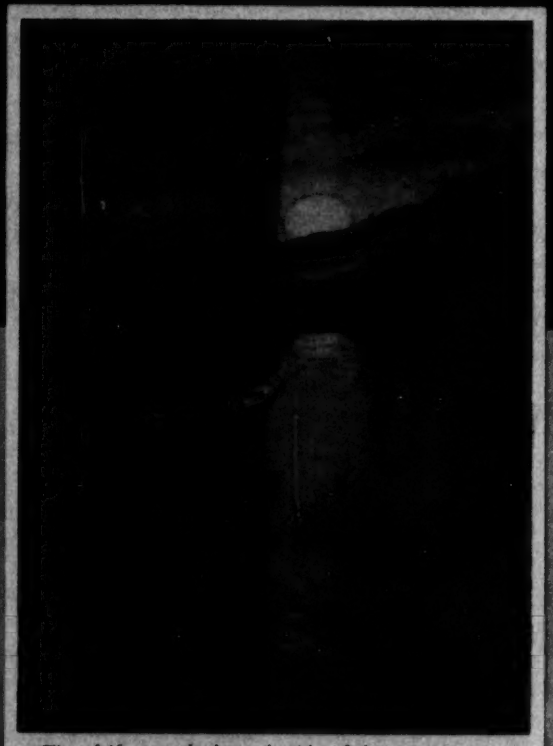

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The effective hypnotic dose in most cases is one or two capsules.

Samples to physicians on request.

Supplied in bottles of 25, 100 and 500 3 grain capsules.

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